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THE TREATMENT OF COMPOUND FRACTURE OF THE SKULL*

A Study of 185 Cases

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INTRODUCTION. Compound fracture of the skull occurs in roughly 10.5 per cent of all cases of skull and brain injury. In a group of approximately 900 head injuries, it is only one per cent less frequent than subdural hematomas and the two together comprise 22 of the 29 per cent of cranial injuries that require operative therapy. Such a large group of patients justify more interest than has been accorded them in the past. It is the purpose of this paper to present figures which will demonstrate that such increased interest pays dividends in the form not only of lowered mortality but also of a great decrease in morbidity.

A review of the literature for the past ten years yields only one paper given over to an adequate exclusive consideration of the subject[7]. This was written from Germany. The authors analyzed 169 cases treated over a period of twenty-three years. Their mortality (41 per cent) and morbidity (11 per cent) rates are high for this reason. The earlier poorly treated cases are not separated from the later ones which were operated upon under modern conditions and by modern methods except in a late result series of posttraumatic epileptics. Here the incidence was 6 per cent in the group treated by a modern closed débridement method as opposed to 12 per cent in a group of about the same size treated by the old open packed wound method. Operation in the first twelve hours with complete mechanical débridement and closure without drainage except where the frontal sinus is involved is urged. Grant[1] on the other hand advises only a modified débridement of the dura and brain only if cerebrospinal fluid is leaking. He drains all wounds. Nelson's Loose Leaf Surgery[10] does not list "compound fractures of the skull" in the index. What text there is on this subject is under "depressed fractures". Homans[9] gives the subject a short paragraph advocating débridement in six hours without drainage. If done later the cortex should be drained. In Lewis' Surgery[11] prompt débridement (within twelve hours) without drainage is advised. Large free fragments of bone are replaced if removed within this time after the accident. Other authors[2, 3, 4, 5, 6, 8] discuss the subject from the point of view of experience with a single case or as one that requires no explanation.

The Mortality Rate. This paper is based on a series of 185 cases that have been collected from the records of the Boston City Hospital. Ninety-six of these were treated on the general surgical services in the thirteen years from July, 1922 to July, 1935. The remaining eighty-nine were treated on the neurosurgical service of the same institution during the five years from July, 1930 to July, 1935. The cases from the general surgical services were treated in a variety of ways. These included no operation, cleaning and sewing the scalp wound only, débridement of scalp wound only with drainage, and débridement of the scalp wound with varying degrees of débridement of the bone and rarely also of the dura but practically never of the brain. No cases were treated until they were out of surgical shock. The diagnosis was usually made by inspection with instrumental palpation through the wound. More recently though, palpation by the operator's gloved finger has replaced this method to a great extent. The operative technique varied greatly with the individual surgeon but it was only rarely that an adequate débridement was performed and even less rarely that a wound was closed without drainage. Where the scalp wound was treated at all on admission it was always shaved, scrubbed and mechanically cleansed at once. Such practices, however, may be fairly taken to represent an average cross section of the general surgical methods in use at the present time. By contrast, on the neurosurgical service all cases were theoretically diagnosed and treated by one standard method to be described in detail below. Any variation, when it occurred, has been recorded against the outcome of the case as a technical error and has been duly considered in evaluating the results.

In the consideration of the comparative mortality of these two groups it became at once ap-

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parent that true treatment mortality could only be ascertained after the elimination of those cases that were so severely injured on admission that all active treatment was out of the question. Therefore the admissions that died without treatment within twenty-four hours have been segregated from the rest of the group. This permits the compilation of mortality rates from two points of view for each series. (Table 1.)

TABLE 1

GENERAL MORTALITY

	Total	Living	Dead	24-Hour Deaths Untreated	Mortality Whole Series	Mortality Without 24-Hour Deaths
Total Series	185	104	81	52	43.7%	21.8%
B. C. H. Series	96	44	52	35	54.1%	27.8%
N. S. Series	89	60	29	17	32.5%	16.6%
Wanke and Weselmann*	169	100	69	49	40.8%	16.6%
Theoretically Possible Figures	89	68	21	17	23.5%	5.5%
Permissible Figures					25.0%	10.0%

*The number of untreated twenty-four hour deaths and the corresponding mortality percentage are only approximate in this group.

Based on the total number in each group the mortality rate in the general surgical series was 54 per cent as opposed to 31 per cent on the neurosurgical service, a difference of 23 per cent. With the cases which died in twenty-four hours eliminated, the difference is still 10.3 per cent lower in the neurosurgical group (17.5 per cent as compared with 27.8 per cent) though these totaled seventy-four cases as opposed to sixty-one and in spite of the fact that no case regardless of condition was refused treatment. It should be pointed out that all diagnoses in both groups that were unconfirmed either by operation or direct examination of the wound were verified in case of death through the courteous coöperation of Dr. Timothy Leary, Medical Examiner of Suffolk County.

The Morbidity Rate. The mortality rate in compound fractures of the skull is only half the story, however. Leaving out of consideration all problems of end-results beyond the actual period of hospitalization, the question of postoperative and postinjury sepsis must still be dealt with. It is well recognized that meningitis, encephalitis, intracranial abscess and healing of the scalp wound by granulation with resultant adherent scars between scalp and cortex, are considered inevitable consequences of this type of injury. These are all in fact the immediate end results of wound infection. The morbidity from sepsis in both series was therefore compared. The figures include only those patients that left the hospital alive as it was felt that these were the only cases in whom an accurate diagnosis of postinjury or postoperative sepsis could be made. (Table 2.) In both groups all wounds were considered septic that did not heal by first intention. In case drainage was inserted only the healing of the wound exclusive of the opening of the drainage tract was considered. The difference of approximately 52 per cent in the operated cases and 30 per cent in the non-operated cases speaks strongly in favor of the standardized method of treatment as a means of reducing the morbidity.

Operative Mortality and Morbidity. Any consideration of a surgical procedure that involves operative interference must also include a study of the mortality and morbidity inherent in it. Again let me emphasize that the figures included herewith concern themselves only with mortality and morbidity during the patients' hospital stay. Table 3 summarizes these figures from both series and also includes the percentage of operated cases. In spite of the fact that more than 70 per cent of the neurosurgical patients were operated upon as opposed to less than one-half of those admitted on the surgical side, the operative mortality of the former is about 10 per cent below the latter, while the morbidity from sepsis has been reduced by more

TABLE 2

ANALYSIS OF MORBIDITY

	Total Treated	Operated Total	Operated Clean	Operated Septic	% Sepsis	Not Operated Total	Not Operated Clean	Not Operated Septic	% Sepsis
Total Series	104	85	58	27	31.7	19	10	9	48.2
B. C. H. Series	44	32	12	20	65.6	12	5	7	58.3
N. S. Series	60	53	46	7	13.2	7	5	2	28.5
Theoretically Possible Figures	60	53	53	0	0.0	7	5	2	28.5

than 50 per cent. It seems fair to conclude therefore that well-conceived operative therapy will not only of itself lower the death rates but will also, if properly carried out, reduce the occurrence of postoperative sepsis from three out of four cases to slightly more than one out of five cases.

TABLE 3

OPERATIVE MORTALITY AND MORBIDITY

	Total Operated	% Operated	Dead	% Mortality	Sepsis	% Sepsis
Total Series	108	58.3%	23	21.4%	48	44.4%
B. C. H. Series	44	45.8%	12	27.2%	33	75 %
N. S. Series	64	71.9%	11	17.4%	15	23.4%
Wanke and Weselmann*	162	95.8%	62	38.2%	17	10.3%
Theoretically Possible Figures	64	71.9%	3	4.6%	1	1.5%
Permissible Figures				10.0%		5.0%

*The figures and percentages of operation and sepsis are only approximate in this group.

Analysis of Deaths. A final test of the justification of a method of treatment and a corollary to the analysis in the preceding paragraph is found in a detailed study of the causes of death. Table 4 gives a tabular view of such figures in these two groups. It is in the main self-explanatory and needs emphasis only in one or two points chiefly because of their bearing on what follows. The first and most important is the fact that in the general surgical services, three cases died because the diagnosis was missed, two from the effects of the compound fracture and one from an extradural clot. The first two did not come to operation at all while the third was operated upon but so incompletely that the source of the clot was not recognized even then. The obvious implication is that the diagnostic methods as used on the general surgical services were inadequate. The second point has to do with immediate postoperative deaths. The majority of these were from surgical shock and occurred within twenty-four hours after the operation while medullary edema at a somewhat later time accounted for the others. The ratio of five to two emphasizes the fact that these patients were operated upon too soon after their injury. The corollary to this is, as pointed out below, that immediate emergency treatment of compound fracture of the skull is not only unnecessary but is actually dangerous. The one death from hemorrhage occurred on the table and resulted from an inadequate appreciation of the dangers inherent in a fracture which crosses a cranial venous sinus. The deaths due to pneumonia and heart failure are self-explanatory.

TABLE 4

ANALYSIS OF DEATHS

		Operated		Not Operated		Causes of Death — Sepsis			Causes of Death — Operative			Causes of Death — Miscellaneous			
	Total Treated	Clean	Septic	Clean	Septic	Meningitis	Brain Abscess	Sepsis	Medullary Edema	Surgical Shock	Hemorrhage	Diagnosis Missed	Extradural Clot	Pneumonia	Heart Failure
B. C. H. Series	17	6	6	2	3	5	2	1		5	1	2	1		
N. S. Series	12	3	8	1		5	3		2					1	1

Treatment. In dealing with any method of treatment of compound fracture of the skull it is essential that certain fundamental principles be recognized and followed. These are both general and specific in nature. In general the operator must always bear in mind that he is dealing not alone with a lesion of the bony covering but that the scalp, the meninges and the brain are all also always involved. If this conception is held firmly in mind the requirements of any method of treatment will be more apparent. Furthermore he must for his own protection locate the bony lesion in relation to the large cranial venous sinuses and also in relation to the frontal air sinuses. This knowledge will prevent him on the one hand from stumbling blindly into a fatal operative hemorrhage and on the other will permit him to place his incision in such a way as to provide for adequate drainage later. As far as specific procedures go the most important is to realize that if a débridement is attempted it should be 100 per cent in type and should not end in a drained wound. Any neces-

sity for the latter is obviated by properly performing the former. Tissue necrosis from too tight stitches must also be carefully avoided. Finally inasmuch as the infection deposited within the wounds at the time of compounding tends to remain static for a period unless new blood and lymph channels are opened for it to spread circumferentially, any cleansing procedures that might facilitate this spreading must of necessity be barred until they can be immediately followed by mechanical excision of the presumably newly infected areas.

All cases treated by the method to be described had at least an open wound of the scalp and pericranium immediately above a fracture of the underlying bone. At the worst the soft tissue injury was a widespread avulsion, the bone was comminuted and depressed, and the dura grossly torn with cortical tissue and cerebrospinal fluid extruded onto the scalp and hair. No wound was considered compounded that had an intact pericranium or one of the more superficial scalp layers, or that did not have at operation a fracture line in the bone beneath the scalp wound.

Preoperative Preparation. On admission to the hospital the patient's blood pressure is recorded and the operator's finger covered with a sterile glove is inserted into the scalp wound and the diagnosis made by palpation only. A dry sterile dressing and bandage is then applied. If the patient is in surgical shock this is treated. It is important to note that at this time no attempt whatsoever is made to clean the wound in any way. The hair is not cut or shaved, no bone fragments or dirt are removed, no washing done or antiseptics applied and only one person allowed to palpate the wound and be responsible for the diagnosis. No variation from this method is permitted. The patient is then sent to the ward and not operated upon for at least twenty-four hours after receipt of the injury whether or not he is in shock during that time. The only permissible therapy in this interval other than that directed toward his shock is to administer hypertonic glucose intravenously or perform a lumbar puncture as a treatment for the associated brain pathology. At the end of this twenty-four hour postinjury period, if the patient is in good condition, he may be operated upon. If there is any doubt about his condition or if he needs to be transported to another locality, a further delay up to thirty-six or forty hours after the accident can be safely permitted. The operation must be performed in any case, however, within forty-eight hours or else postponed for three to six months. While there is a theoretical possibility that a complicating bleeding sinus or meningeal artery may rarely force operative interference in the first twenty-four hours after injury, my experience has been that such cases are obviously moribund on admission and eliminate themselves from among the operative possibilities by dying very promptly. This preoperative waiting period may be put to good use to move the patient to the most favorable hospital from the point of view of equipment; to determine, if necessary by x-ray, any involvement of the frontal air sinuses; and particularly to arrange for the attendance of suitable donors for the provision of blood should transfusion prove necessary during or after operation.

Operation. His suitability for operation having been ascertained, the patient is placed on the operating table and there for the first time the original bandage and dressing covering the wound is removed. Depending on the degree of coma, the severity and extent of the injury, and the coöperation of the patient, either local or general anesthesia may be employed. If possible a basal anesthesia from avertin is first induced in either case. If a general anesthetic is to be used in addition, I prefer ether by the drop method. The entire scalp is next clipped and shaved and the head well scrubbed with soap and water on gauze, followed by repeated wipings with alcohol and subsequent scrubbing with ether. Care is taken to keep the solutions out of the open wound. The area is then painted with 1 per cent iodine solution, the wound itself being included in this. If local anesthesia is chosen, 1 per cent novocain with two drops of adrenalin to the ounce of solution is employed. This is injected in a circle large enough to include in addition to the wound, enough adjoining tissue so that a plastic type of closure can be easily carried out if necessary. The incision is then outlined in such a manner as to surround the wound and permit the necessary excision of its edge. The amount to be excised depends upon the length of time intervening between injury and operation and the amount of contusion of the wound edges. If there is more than one laceration, care should be taken so to place the operative incisions that they will not parallel each other. Necrosis of the intervening scalp will ensue if this precaution is not observed. If the wound to be excised is so large as to require a mobilization of the scalp to close it, an arm should be extended laterally from some point in its middle one-third into the largest area of undamaged scalp. This lateral arm *must* leave at such an angle as to form a Y shaped incision, however, and never at right angles with a resultant T shape. This latter T shape provides no extra mobilization, an advantage which accrues only from the Y shape. After appropriate draping the original wound of the scalp and underlying layers including the pericranium is débrided "en bloc" and these instruments discarded. The rest of the incision is then completed and hemostasis obtained by spaced hemostats placed on the galea and turned back across the edge of scalp. The fracture is thus exposed but only the compounded part débrided. In the

majority of cases this is accomplished by drilling a hole in clean bone to one side of the fracture line and with this as a starting and ending point and a De Vilbiss rongeur as an instrument, excising the bone edges in either one or two pieces. An exception must be made to this method when working in the neighborhood of any of the venous sinuses or the supraorbital ridge. This will be considered below. Excision of the bone will expose the potentially infected dura which, if untorn, should not be interfered with further. If intradural hemorrhage is suspected, however, and further exploration is desired, enough more bone can be removed so as to expose clean dura. The membrane can then be opened for exploration at this point. If the dura has been torn, the torn edges are excised and the opening extended sufficiently to expose satisfactorily the underlying cortex. It is here that the inexperienced or poorly equipped operator usually fails to complete his débridement, in that he neglects to deal with the contusion or laceration of the exposed brain. This is usually due to the mistaken belief that removal of the surface clot will permit more hemorrhage which he will be unable to control, or that he will remove viable brain tissue and thus extend the scope of the original injury. Neither of these conceptions is correct and both are harmful in that they, of necessity, leave behind an incarcerated mass of culture media which probably has already been inoculated. If the latter happens to be true, a cortical abscess is the inevitable result. Such a complication can be prevented by completing the débridement through the excision of the contused cortical tissue with its associated clots and bacteria. This should be thorough regardless of the amount of apparent resultant damage, and can be successfully and harmlessly carried out by the use of a suction apparatus. Ordinary suction will not remove normal brain tissue but does excise all the damaged tissue. Secondary cortical bleeding is largely venous and either stops of itself with the subsidence of the associated venous congestion or can be controlled by a small muscle implant on the open end of the bleeding vein. Cortical arterial bleeding is rare but can be easily handled by the application of a silver clip to the artery. Neither clip nor muscle graft will act as a foreign body providing the débridement is complete and future sepsis locally has been thus eliminated. This completes the final step of the débridement. The closure starts with the dura and should be tight if possible. If this cannot be done, a loosening parallel incision should be made laterally. By thus permitting the closure of the original dural wound, that part of the cortex that will come to lie beneath the scalp scar can be better protected. This will minimize later adhesions. In large defects a transplant of fascia lata can be made at this time providing the fracture has involved neither the frontal air sinus nor the supraorbital ridge. No attempt is made to close the bony opening. The scalp is then approximated by two layers of interrupted sutures of No. 7 silk. The wound should be closed from the periphery toward the centre and the edges must be brought together without tension. To accomplish this the scalp and galea may be widely undermined in all directions by separating the galea from the pericranium. This together with the Y shaped incision will permit considerable sliding and the closure of very large defects. No drainage of any sort is used except as noted below. As a final safeguard and in recognition of the fact that a skin suture loose at the time of tying becomes so tight as to cause necrosis after the postoperative subcutaneous edema has had a chance to develop, the areas of skin on either side of the suture lines are extensively scarified with short multiple incisions deep enough to cause bleeding. Particular attention is paid to the apices of any triangular shaped flaps. The wound is then covered with silver foil, a dry sterile gauze dressing and a bandage. Fifty per cent glucose solution in 100 cc. amounts and repeated blood transfusions in 125-250 cc. amounts are used intravenously to combat surgical shock or hemorrhage either during or after the operation. Caffein sodium benzoate is given in 7½ gr. doses, also intravenously, to stimulate either a failing circulation or to correct respiratory embarrassment of central origin. For the rest, well-recognized general surgical principles govern the postoperative care. The stitches are removed from the skin on the third day and the head well cleaned with soap and water on the tenth day.

Exceptions. No exceptions to the above technique are knowingly permitted unless one of three complications exists. The first of these has to do with the débridement of the bone when the fracture line overlies a cranial venous sinus. Here the use of the De Vilbiss rongeur is too likely to tear into the sinus and the compounded bone is removed piece by piece with a biting rongeur. At the same time preparation is made to obtain quickly a piece of muscle from the patient's calf to use as a postage stamp graft to close any opening in the sinus. This is the only practical method available in these cases for dealing with what may prove otherwise to be a fatal hemorrhage. Attempts to apply hemostats will tear other holes and packing either will not control the hemorrhage or will lead to later wound infection with its complicating aftermaths.

The second exception also has to do with the débridement of the bone and occurs when part of the compounded fracture line involves the supraorbital ridge, lateral to the frontal sinus. Under these conditions the débridement must be intentionally incomplete and this portion of the involved bone left in place. In the face of this incompleteness the wound must be drained,

FIGURE 1. To show Mosher drain in place three weeks after operation. Drain removed eleven days later. Patient discharged with wound completely healed in another three weeks.

FIGURE 2. To show condition of wound after drainage with Mosher drain, at discharge nine weeks postoperative. No fistula from sinus. Note Y shaped scalp incision.

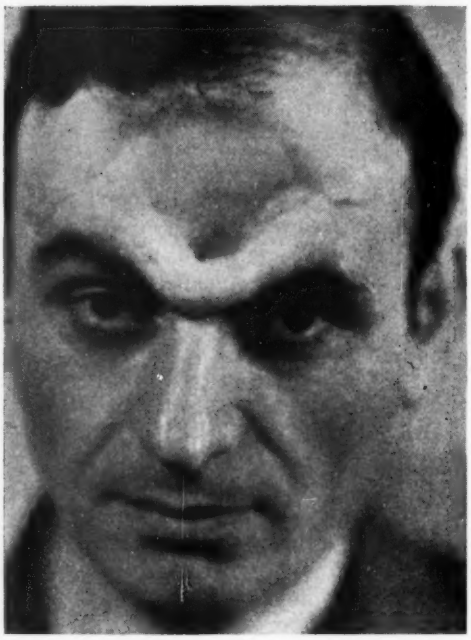

FIGURE 3. To show condition of wound after drainage with Mosher drain and with a fistula into sinus one year after operation. Both sinuses involved, the fistula is slowly closing in of itself.

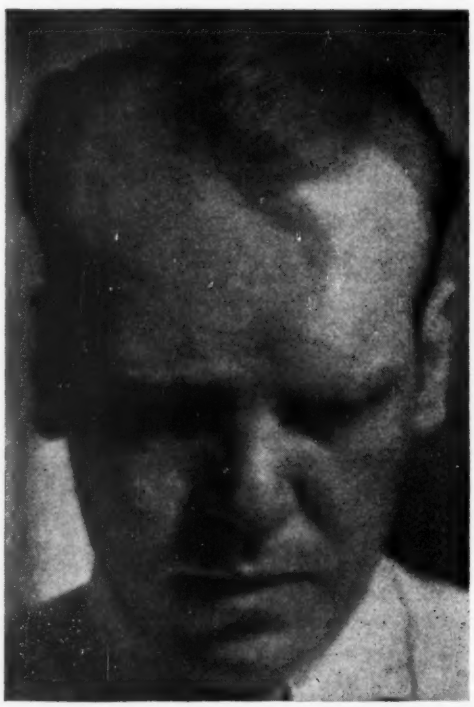

FIGURE 4. To show end-result of extensive injury to both frontal sinuses, the frontal lobes and the frontal bone. Operated four years, three months ago. Mosher drain used. Fistula to sinus closed by plastic operations. Patient now symptom free and leading a normal life.

preferably with rubber tissue and from two to four days. Any other and more ideal procedure, while it will lessen the chance of postoperative infection, subjects the patient to an unsightly and uncomfortable deformity. With the supraorbital ridge gone all support for the frontal lobe when the individual is bending forward has been removed.

The third exception is more important and merits more detailed consideration. It again has to do with incomplete débridement of the compounded bone. It occurs in those cases in whom the fracture line extends into the frontal air sinus. By a fracture of either the anterior or posterior wall of the frontal sinus especially if there is a gross tear of the mucous membrane a communication is established between the cranial cavity and the nose. This is, moreover, by way of an already infected portal. Because of anatomic peculiarities as well as the mechanical impossibility of closing off the nose, a complete débridement is out of the question in these cases. Drainage of the wound therefore becomes essential. Moreover where the dura has been torn and the brain lacerated, ordinary drainage with rubber tissue or gauze is inadequate. This is due to the tendency of the infected brain to fungate and to enclose within itself the infecting agent. To overcome this difficulty I have used the conical shaped "Mosher" wire screen drain. The lower end is left at the depth of the cortical excision and the upper end at the level of the skin with the dura sutured to it with three interrupted sutures. By thorough curettage of the inside of the drain once a day, drainage is provided for the brain tissue, exuberant granulations are kept down and, after cutting the dural stitches at the end of a week, the gradual filling of the tract with scar tissue will force the drain out at the optimum rate. Provision is also made for drainage of the frontal sinus through the operative wound. The accompanying photographs illustrate the different stages of the healing of such a wound. (Figures 1-4.) This series from the neurosurgical service includes ten such cases. All were operated upon with four dying following operation. Of the six that lived, five had no complicating sepsis. The sixth recovered from a secondary cortical abscess despite the fact that he was not operated upon until six days after his injury. The cause of death of the remaining four will be considered in detail with the rest of the fatalities.

Discussion. The method of treatment as described above presents certain obvious points that are susceptible of discussion. One of these is the amount of bone to be removed at operation and the advisability of attempting to replace large fragments in order to prevent deformities. As far as the removal of the bone goes, I believe that all bone that may have been contaminated at the time of the injury by having come in contact with septic material should be removed. In these cases it is not primarily a problem of the best looking end-result but one of the mechanical removal of material contaminated by bacteria. With a simple linear fracture underneath an incised wound of the scalp, this presents no difficulties. It is obvious that the edges of the fracture line are the parts that must come out. Where one is dealing with the more complicated situation brought about by the comminution of the bone associated with any degree of avulsion of the scalp it is a different matter, however. Here the best end-result is reached by the removal of all bone that has been separated from its pericranium at the time of the injury. If, as sometimes happens, there has been an extensive separation of the galea and pericranium as well, this contaminated pericranium should also be removed. If this exposed bone is fragmented, it will all have to be sacrificed. If, on the other hand, some of the fragments are only partly denuded of their attachment to the scalp, the denuded part only needs to be removed. I am opposed to the replacement of large free fragments of bone in compounded fractures of the skull. If it must be done, I believe that the fragment should first be sterilized by boiling but I have had no personal experience with replacement after any method of sterilization. It seems to me that such a procedure is bound to carry a high percentage of failures and a failure in this type of case means postoperative sepsis. No matter how careful the débridement, the sterilization of the wound is only relative. However, if no further demand is made on the reparative processes of the body, such a wound will heal by first intention. If, on the other hand, a large foreign body, such as a fragment of bone which has been completely deprived of its blood supply, has to be cared for in addition, the percentage of postoperative infection is bound to rise. Attention should be called also to the problem presented by the fracture which is compounded through the scalp at its posterior end and in addition has its anterior end opening into a frontal air sinus. This has only recently been illustrated by a personal case which is not one of those included in this series. The outcome was a fatality from septicopyemia in spite of a technically adequate operation in the part compounded through the scalp. The infectious process was found to have extended backward from the frontal sinus along the uncompounded portion of the fracture line. In future I believe that I shall make provision for drainage of the frontal sinus end of the fracture in such a case.

Attempts to culture the wounds at the time of operation have been made but the results have been too variable and the cultures too few to justify any conclusions on this basis.

The importance of following in detail the

operative method as described is emphasized by the accompanying table 5. This lists the significant data in all the cases of death and sepsis in the neurosurgical group regardless of the method of treatment. Two deaths, one due to sepsis, occurred after operations that, as far as could be told, were technically perfect in both cases. The uncomplicated death was due to pneumonia in fifteen days after admission. That due to sepsis occurred in twenty-one days from a streptococcus hemolyticus meningitis in which the bacteria were demonstrated in the cerebrospinal fluid twelve hours after the operation. Other fatalities included two which were the result of an operation performed while the patient was possibly still in surgical shock. They were done twenty-eight and nineteen hours after injury respectively. All other deaths were secondary to infection except one in which an incidental heart failure was the cause.

The greatest single cause of postoperative sepsis was some type of major technical error in the performance of the operation. This occurred as the sole cause ten times, and was a factor in two other septic cases. This is 60 per cent of the entire group. The errors varied from parallel scalp incisions, and packing a wound to control bleeding, to failure to débride both bone and brain. Half of the twelve died and the other six had an average hospitalization of thirty-nine days, the maximum being seventy-eight and the minimum twenty-three. Improper handling of the wound on admission caused no deaths and no major sepsis but was associated with two infected scalp wounds in neither of which was any operative procedure carried out. Operations performed after the forty-eight hour period were the only cause of sepsis in four cases, three of whom died. The postponement was for nine days, four days, seventy-two hours and fifty-five hours after the injury respectively.

It is obvious that technical errors in operating are subject to correction and that operative interference at too late a time is inexcusable. It is theoretically possible therefore to calculate ideal mortality and morbidity rates for the method of treating compound fractures of the skull as described. For this purpose it is reasonable to consider eight deaths as preventable (all having been operated) and sixteen cases of sepsis as preventable, fourteen of them having been partially débrided. When revised in this manner, this neurosurgical series has a gross mortality of 23.5 per cent in a group of eighty-nine cases and including all cases dying untreated within twenty-four hours of admission. If these latter are excluded the theoretical mortality of treatable compound fractures of the skull in this series of seventy-two cases drops to 5.5 per cent. The operative mortality and morbidity in the sixty-four débrided compound fractures of the skull takes an equally striking drop. Here the mortality rate drops from 17.4 per cent to 4.6 per cent and the morbidity from sepsis from 23.4 per cent to 1.5 per cent.

CONCLUSIONS

1. From a series of ninety-six compound fractures of the skull admitted to the general surgical services of the Boston City Hospital during thirteen years, and lacking other confirmatory data it is suggested that the mortality of this injury as treated by the general surgeon has a gross figure of 54.1 per cent and a net figure of 27.8 per cent.

2. From a series of eighty-nine similar cases admitted to the neurosurgical service of the Boston City Hospital during five years and treated by a standardized method the actual gross mortality was 32.5 per cent and the net 16.6 per cent. It should be possible to reduce these figures further to the neighborhood of 23.5 per cent and 5.5 per cent.

3. The morbidity from sepsis in compound fractures of the skull when treated by general surgical methods ranges from a total of 75 per cent, through 65.6 per cent in the living operated cases to 58.3 per cent in the living unoperated ones.

4. The morbidity from sepsis in compound fractures of the skull when treated by the special method in use in the Boston City Hospital neurosurgical service ranges from a total of 23.4 per cent, through 13.2 per cent in the living operated cases to 28.5 per cent in the living nonoperated ones. It should be possible to reduce these figures further to the neighborhood of a total of 1.5 per cent, through 0 per cent in the living operated cases but with the same 28.5 per cent in the living unoperated ones.

5. A method of treating compound fractures of the skull is described. Emphasis is placed on the care of the wound on admission, the length of the preoperative interval, the method and completeness of the débridement and the requirements of closure of the wound.

6. The cases that either died or developed sepsis in this neurosurgical service series are discussed in detail from the point of view of method and efficiency of treatment.

7. The maximum permissible mortality rate in compound fractures of the skull should be grossly not over 25 per cent. If the cases that died untreated within the first twenty-four hours of admission to the hospital are eliminated the mortality rate should not be over 10 per cent.

8. The maximum permissible operative mortality rate in treated compound fractures of the skull should not be over 10 per cent.

9. The maximum permissible morbidity rate from sepsis in treated compound fractures of the skull should be not over 5 per cent.

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TABLE 5

ANALYSIS OF FATAL AND SEPTIC CASES IN NEUROLOGICAL SE

Age	Location of Fracture	Type of Treatment		Technical Errors in					Drainage		Complications		
					Débridement of								
		Op.	Non-op.	Admission Dressing	Scalp	Bone	Brain	Suture + Scarification	Yes	No	Venous Sinus	Supra-Orbital Ridge	Frontal
6 yrs.	Frontal + Temporal	√							√			√	
32 yrs.	Frontal Bone + Sinus	√		√		√			√				
75 yrs.	Frontal Bone + Sinus Mult. Injuries	√							√				
24 yrs.	Frontal Bone Cribriform Plate Mult. Injuries	√		√		√	√		√				
36 yrs.	Frontal Bone + Sinus	√				√	√			√			
29 yrs.	Frontal Bone + Sinus	√		√		√	√		√				
58 yrs.	Frontal Bone	√		√	√		√			√			
8 yrs.	Frontal Bone Mult. Injuries	√								√			
20 yrs.	Temporal Bone	√			√				√				
60 yrs. +	Vertex-Biparietal	√		√					Pack		√		
13 yrs.	Occiput	√				√			Pack		√		
7 yrs.	Frontal Bone	√					/		√				
58 yrs.	Frontal Bone	√				√	√		√				
54 yrs.	Parietal Bone	√			√					√			
11 yrs.	Parietal Bone	√						√		√			
36 yrs.	Parietal Bone	√						√		√			
53 yrs.	Parietal Bone	√						√	√				
46 yrs.	Frontal Bone	√		√						√			
15 yrs.	Parietal Bone		√	√									
32 yrs.	Frontal Bone		√	√									
69 yrs.	Occiput		√	√									

TABLE 5

SEPTIC CASES IN NEUROLOGICAL SERIES

Drainage		Complications			Pre-Operative Interval	Cause of Death or Sepsis	Length of Stay in Hospital		Chief Cause o
es	No	Venous Sinus	Supra-Orbital Ridge	Frontal Sinus			Dead	Living	Death or Sepsi
√			√		28 hrs.	Medullary Edema	24 hrs.		Operated too soo
				√	55 hrs.	Meningitis at Operation	6 days		Operation too la
				√	40 hrs.	Pneumonia	15 days		O. K.
				√	14 hrs.	Medullary Edema	48 hrs.		Operation too so
	√			√	8 hrs.	Frontal Abscess	23 days		Technical Error
				√	9 days	Frontal Abscess Meningitis	26 days		Operation too lat
	√				24 hrs.	Cortical Abscess	4 days		Technical Error
	√				24 hrs.	Strep. Meningitis in 12 hrs.	21 days		O. K.
√					4 days	Cortical Abscess Strep. Meningitis	30 days		Operation too lat
ck		√			24 hrs.	Meningitis	6 days		Technical Error
ck		√			24 hrs.	Meningitis	2 days		Technical Error
					5 hrs.	Granulating Scalp Wound		30 days	Technical Error
					6 hrs.	Cortical Abscess		78 days	Technical Error
	√				24 hrs.	Necrosis of Scalp		30 days	Technical Error
	√				36 hrs.	Granulating Scalp Wound		30 days	Technical Error
	√				24 hrs.	Granulating Scalp Wound		23 days	Technical Error
					24 hrs.	Granulating Scalp Wound		44 days	Technical Error
	√				72 hrs.	Granulating Scalp Wound		30 days	Operation too lat
						Infected Scalp Wound		19 days	Sutured on Admission
						Granulating Scalp Wound		13 days	Wound packed on Admission
						Heart Failure	6 days		Incidental

f Cause of h or Sepsis
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REPORT OF A CASE OF LEAD ENCEPHALITIS DUE TO TRI-ETHYL LEAD*

BY KARL M. BOWMAN, M.D.,† AND PAUL M. HOWARD, M.D.†

ENCEPHALITIS due to either tetraethyl or tri-ethyl lead is quite rare and very few cases have been reported since the first group of about seventy cases in 1924 and 1925. It seems worth while, therefore, to present a case of tri-ethyl lead poisoning.

The patient was a white, married male of forty years. He was sent in to the Boston Psychopathic Hospital on September 12, 1934 by Dr. Joseph Aub, who had made a tentative diagnosis of encephalitis from tetraethyl lead.

On admission, the patient was disoriented, confused and had marked disturbance of memory for both recent and remote events with some tendency to confabulation. He could remember nothing that had happened during the past year, could not give a correct chronological history of his past life, and could not recall an address or name after one minute; he gave his age as about thirty-seven. There was no disturbance of mood although he seemed somewhat restless, no special trends were elicited. There was no evidence of delusions or hallucinations, but a few days later he apparently thought that he saw insects. There was no insight, the patient claiming that he was not ill and that his mental faculties were not impaired. The mental picture was that of an impersonal organic psychosis.

The physical examination showed a white male, five ft., ten in. tall; weight 147 lbs., with evidence of recent loss. He appeared seriously ill. The heart, lungs, abdomen and extremities were normal. The throat was not injected although the patient complained of a sore throat and cough. There was no evidence of dehydration. Blood pressure was 110/80; the pulse rate was 80. The neurological examination showed a coarse tremor of the hands, arms and head, and fine tremors about the mouth. The deep reflexes were equal and active. There were none of the classical signs of lead poisoning such as lead line, wrist drop or muscular weakness, colic, anemia or stippling of red blood cells.

The laboratory findings showed variable traces of sugar in the urine, with some acetone and the slightest possible trace of albumen. Microscopic examination was negative. At the same time, the blood showed a nonprotein nitrogen of 48.4 mgm. per 100 cc. and a sugar of 141 mgm. per 100 cc. The spinal fluid sugar was 110 mgm. per 100 cc. These findings cleared up within a few days and there was no further evidence of faulty sugar metabolism. The other laboratory findings were negative at this time. The spinal fluid findings including dynamics except for the increased sugar were normal.

Specimens were sent to Dr. Robert Kehoe of the University of Cincinnati Medical School and Dr. Lawrence T. Fairhall of the Harvard Medical School. Dr. Kehoe reported that on September 29 the blood showed 0.15 mgm. of lead per 100 Gm. of blood and that the urine showed 1.13 mgm. of lead per liter of urine by spectrographic method. On October 30 he reported that the blood showed 0.13 mgm. of lead per 100 Gm. of blood. However, this test tube had broken in transit so the results were not considered accurate. A urine specimen at the same date showed 0.10 mgm. of lead per liter of urine. A third specimen of blood collected November 16 showed 0.07 mgm. of lead per 100 Gm. of blood. A urine specimen collected at the same time showed 0.05 mgm. of lead per liter of urine.

Dr. Fairhall reported that two specimens of spinal fluid were questionable for lead, that a twenty-four hour specimen of feces on September 18 contained 0.3 mgm. of lead, a second twenty-four hour specimen on September 24 contained 0.77 mgm. of lead and a third twenty-four hour specimen of September 28 contained 0.77 mgm. of lead, that a urine specimen of 2200 cc. on September 26 contained 1.4 mgm. of lead and a second specimen of 4000 cc. of September 28 contained 0.19 mgm. of lead. In these specimens the lead was additionally separated as sulphate and identified positively as lead by microchemical analysis.

The history shows that the patient was of Scotch English descent, with no family history of mental or nervous disease or alcoholism. Birth and early development were normal. He finished high school and took a night course in accounting. He held various jobs and was considered a conscientious and efficient worker. For the past year he had worked for an oil company and recently he had worked at cleaning gasoline tanks. In 1923 he had an appendectomy and in 1925 a light attack of pneumonia. Otherwise there was nothing of significance in the medical history. He was married in 1921. In personality the patient was described as cheerful, energetic, industrious, popular, and even-tempered.

The exact details leading up to his illness were not entirely clear. The patient was employed cleaning out gasoline storage tanks. He wore an asbestos suit and a gas mask connected with a pipe line to an oxygen supply. His duty was to remove the crust or scale from the sides of the tank after they had been emptied. Between August 20 and September 4, he entered nine gasoline tanks. About August 21 to 23, he complained that he could not smoke on account of burning in the eyes, nose and throat. He also said that there was something wrong with the air supply in his gas mask. On August 30 headache and insomnia developed. He began to lose weight rapidly and complained of great body fatigue. On September 5 he collapsed while at work, was quite pale but managed to stagger about. He was

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taken to a general hospital, where he was kept until the next day and then sent home. On September 6 he was perspiring profusely, had headache, and again collapsed. On September 7 he was weak and confused, and showed mental symptoms. His insomnia had continued throughout this period. On September 8 he was very restless. On September 9 his restlessness continued and he showed marked memory impairment. This picture continued until the time of his admission to the Boston Psychopathic Hospital on September 12.

After admission to this hospital the patient grew worse rapidly. He began sweating frequently and profusely. He would lie on his bed, rolling his head back and forth in a fashion sometimes seen in rickets. The temperature which was normal on admission rose to 102° and the white blood count which was 6,400 on admission rose to 18,000. He became practically comatose with absent reflexes. There was an area of hyperesthesia to light touch over the lumbar region. Deep pressure, however, seemed to cause no pain. There was vague generalized abdominal tenderness. The only signs elicited in the chest were that the breath sounds were heard better on the left side than the right. This condition soon cleared up, the fever fell by lysis, the confusion disappeared and memory improved.

On October 5 he was transferred to a convalescent ward. It was noted at this time that the temperature had been normal for a week and that the white blood count was normal. On October 15 it was noted that he was improving steadily. His memory appeared fairly good for past events, but quite poor for recent events. He could not remember having worked at any time cleaning gasoline tanks. A special memory test was done on this date, using the Wells memory examination. It was noted that the patient was very coöperative, that he worked with excellent effort and with considerable interest. Memory for recent events was especially poor, scoring below average and, in two items, below the normal limits. Memory for old material was good. These findings were considered consistent with the clinical history of the case and were felt to represent the type of memory defect one would expect in a case of this type.

About October 20 the patient developed a cough and was put to bed. It was noted at this time that he had been quiet and coöperative, that he seemed alert and cheerful, talking freely with others and being quite sociable. No delusions or hallucinations were present. He seemed definitely confused about events in his past life and could not get the order of his occupations correctly. He developed many more physical symptoms from this time on. By October 26 it was noted that he had a temperature of 101.8°, skin was moist; there was considerable cough; inguinal and epitrochlear adenopathy was noted. There was slight tenderness over the epigastrium.

On October 29 a blood examination showed a hemoglobin of 76 per cent, Sahli. The red blood cells were 3,580,000; the white blood cells 19,000, 70 per cent being polymorphonuclears; the smear was normal. On October 30 an x-ray of the chest was reported by Dr. W. K. Coffin to indicate marked pathology in the chest, suggesting bronchopneumonia, pneumonitis, abscess without cavity, or even neoplasm.

He was seen on November 3 by Dr. S. William Marlowe who noted that there was marked inequality of chest expansion, the right side lagging and not expanding so much as the left; the right supraclavicular fossa was deeper than the left. The heart was not enlarged nor displaced. Posteriorly there was dullness at the right apex, with no alterations in breath sounds or vocal fremitus. After coughing, a few râles were heard at the right apex. No other findings were made out. Tuberculosis was considered the most likely explanation and repeated sputum examinations were done which were negative.

By November 8 the temperature had dropped to 99° and remained normal after that time. The blood examination on that date showed a hemoglobin of 65 per cent, Sahli. The red blood cells were 3,800,000; the white blood cells 22,800, 78 per cent being polymorphonuclears; the smear was normal. The

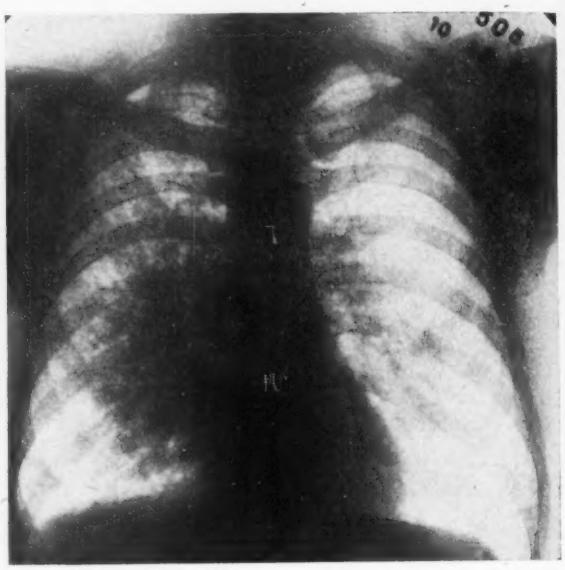

FIGURE 1. Film No. I. X-Ray Report. There is an extremely dense, extensive and mottled, and indefinitely bounded shadow surrounding and radiating from the right hilum and occupying an area the size of a large flattened orange, with bands and striae prolonged raggedly outward and upward to the axilla. In the left side, though, a somewhat similar, but far less dense shadow is seen. Heart negative. Findings suggest a distribution, largely peribronchial, though there is some parenchymal involvement. Probably not Tbc. but it cannot be altogether excluded: bronchopneumonia: pneumonitis: abscess without cavity: possibly even neoplasm and syphilis are to be considered. W. K. Coffin. 10/31/34.

patient's physical condition improved steadily from this time on. He was given iron and ammonium citrate for the anemia and by November 24 the hemoglobin was 80 per cent, Sahli. The red blood cells were 5,010,000; the white blood cells, however, were 20,500, 72 per cent being polymorphonuclears and the smear normal. The urine had been essentially negative during this period.

Two further x-rays of the chest, an oblique chest made on November 13, and a six foot chest plate made on November 22 showed a less intense shadow in the right lung near the hilum, with slight lessening of density in some areas in the right lung. No decided clearing up was noted. These three x-ray plates were examined by Dr. M. C. Sosman of the Peter Bent Brigham Hospital who reported as follows:

"I think it is a very difficult series of plates to read, and in fact cannot tell exactly what this process is.

"It is probably an inflammatory process. I do not think it is tuberculosis or malignancy. The regression shown between the two series of plates is against malignancy. It is possibly a fungus infection and I suggest culture for this. There is something of the appearance of a pneumoconiosis. Was he in a dusty trade? Very possibly it is a bronchopneumonic type of pathology. There is a possible connection between this and his recent exposure to tetraethyl lead."

On November 9 a second memory test showed definite improvement, the memory quotient having risen from ninety-two per cent on, the first test to 107 per cent. From this time on convalescence has been gradual with no remissions.

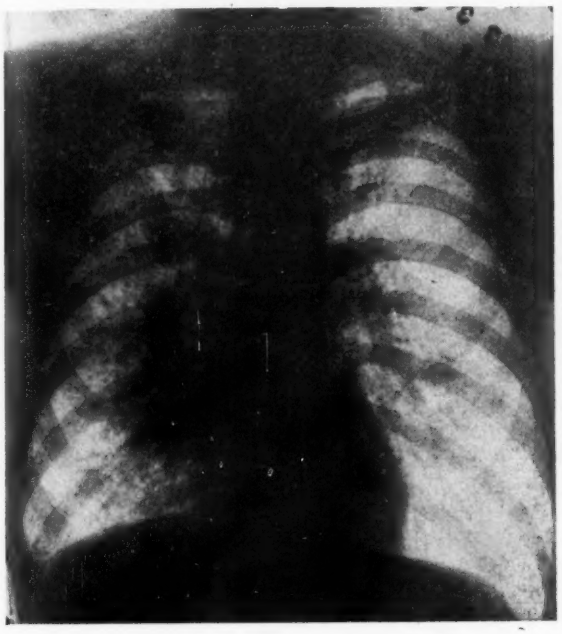

FIGURE 2. Film No. II. X-Ray Report. There is the same general appearance as before, but the intense shadow in the right lung near the hilum is somewhat less dense than before, and apparently there is a somewhat wider diffusion of the clouding both right and left. W. K. Coffin. 11/15/34.

On November 27 it was noted that the patient continued afebrile, that there were no night sweats, but that there was considerable cough and sputum. The right chest showed dullness posteriorly; breath sounds were heard better on the left side

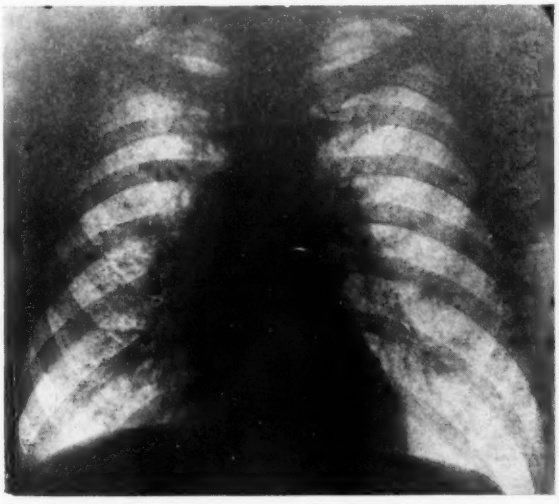

FIGURE 3. Film No. VI. X-Ray Report. Marked improvement since last time. The individual bronchial markings are now quite easily traceable and much of the parenchymal infiltration has cleared. W. K. Coffin. 1/10/35.

than the right anteriorly. There seemed general improvement both physically and mentally.

The patient's weight curve is of some interest: On admission September 12 he weighed 142 pounds. There was a drop to 132¼ pounds by October 5; following this there was a slight gain in weight, so that on October 19 it was 135 pounds. With the acute illness at this time the weight dropped to 125 pounds on November 23. Following this there was a steady rise so that on December 7 it was 130¾ pounds; December 23, 141 pounds; January 4, 148½ pounds; January 31, 157½ pounds.

There has been steady mental and physical improvement. A third memory test on January 26 showed a memory quotient of 118 per cent. The general physical and laboratory examination from examinations made during the last week in January were essentially negative. The urine showed a one plus sugar, but the blood sugar was 97 milligrams per 100 cc. of blood. It was also noted that there were ten white blood cells per high power field in the urine with some clumps which suggested that there might be some pyelitis. Temperature, however, was quite normal and there was no localized tenderness.

An x-ray of the chest on December 8 showed definite improvement of both lungs although there was still a great deal of infiltration, particularly on the right. Three more x-rays taken on January 7, 16 and February 9 show further improvement.

The treatment in this case consisted of forcing fluids and the use of light metals such as calcium and magnesium. It would seem that this patient entered the hospital with a lead encephalitis due to the inhalation of tri-ethyl lead. The reason for assuming that it was due to tri-ethyl rather than tetraethyl is a personal communication from Dr. Robert A. Kehoe, which is as follows:

"I am taking the liberty of discussing briefly one point in the case about which there seems to be some doubt, or perhaps misunderstanding. It seems to be your idea that appreciable quantities of tetraethyl lead may remain on the sides of gasoline storage tanks, and that workmen may be exposed both to this tetraethyl lead and to its decomposition products. This is not quite the situation and I should like to describe, therefore, what happens inside these storage tanks.

"First let me point out to you that the evidence adequately demonstrates measurable amounts of tetraethyl lead (in the employed concentrations) are not absorbed out of gasoline by human beings within tanks or outside of them, either through skin contact or inhalation. Thus the question of tetraethyl lead does not enter into the problem as such under conditions in which gasoline containing tetraethyl lead in the commercial concentrations is involved. The following conditions must be pictured. Gasoline containing tetraethyl lead is allowed to stand for comparatively long periods of time (years) in storage tanks which have a layer of water at the bottom. The decomposition of the tetraethyl lead, which takes place mainly at the surface in the absence of light, results in the formation of finely divided tri-ethyl lead compounds which are only slightly soluble in gasoline, and in water. This finely divided material slowly separates out and small quantities of it are deposited in the scale on the sides of the tank.

Most of it, however, settles to the bottom of the tank, passing through both the gasoline and water layer, and is deposited along with débris from the sides of the tank in the form of a sludge. This entire process is so slow as to be insignificant over short periods of time but the tanks often go uncleaned for years, during which time they are used for the storage of one batch after another of gasoline. Thus after one or more years, there may be a significant amount of material on the bottom of the tank, which is finely divided, and which is relatively high in its lead content, such lead being in part (apparently up to as much as two per cent in our observations) composed of tri-ethyl lead compounds. There is also a measurable quantity of tri-ethyl lead compounds in the rust and scale on the tank. There are only traces of tetraethyl lead since tetraethyl lead does not separate out of its true solution in the gasoline.

"When such a tank is cleaned, if it is emptied out or steamed out and then allowed to air until the sides are dry, and the sludge has been partially dried, any attempts to remove either the scale or the sludge in this dry state result in the dispersion into the atmosphere of finely divided material including tri-ethyl lead salts. These salts make themselves known by their irritating effect upon the eyes and mucous membranes of the nose and throat. They even produce some burning and stinging of the skin. They are absorbed from the pulmonary surfaces with much greater rapidity than most inorganic lead compounds, and due to their chemical properties they are deposited in the tissues of the body in much the same fashion as tetraethyl lead. Their physiological effect is like that of tetraethyl lead. They produce, therefore, a fulminating type of lead intoxication with predominant central nervous system symptoms. Coincident with the rapidity of their absorption, they are apparently excreted somewhat more rapidly than most lead compounds. As a consequence, in both animals and men, lead excretion reaches a very high level shortly after exposure, and falls off much more rapidly than is the case with the more usual lead compounds.

"I have gone to some length to explain this matter because it seems quite clear to me that unless some unusual circumstance occurred, the explanation of the illness of Mr. W. is that he was exposed to such materials in a partially dry state, and either had an unsatisfactory mask or did not make use of it continuously during the period in which he worked.

"This is the only situation I know in which significant amounts of any lead compound separate out of gasoline containing tetraethyl lead, and it is of importance because it provides an occupational hazard at this one specific point. We have investigated the extent of the deposition which occurs in tank cars used for the transportation of leaded gasoline. Here the situation is different not only because of the rapidity of turn-over of the contents, but also because there is no water layer in tank cars to act as a continuous means of separating out the tri-ethyl lead compounds. The tank cars, therefore, are found to contain only insignificant traces of these compounds."

SUMMARY

It is well recognized that tetraethyl lead may produce encephalitis and that mental symptoms form a prominent part of the picture. In fact, when the first series of cases were reported in New Jersey in 1924, ethyl gasoline was called "looney" gas. The symptoms in order of frequency are as follows: Insomnia, lowered blood pressure, subnormal temperature, anorexia, nausea, bodily weakness, abdominal cramps, unaccustomed or annoying dreams, decided loss of weight, slow pulse, headache and tremors. Kehoe[2] reported a series of cases in 1925 in which he emphasized that the clinical picture is different from that usually seen in lead poisoning. He stated that stippling and lead line occur only infrequently and not early in the disease, that the mode of entrance was by inhalation or directly through the skin. He stated that in some cases the mental picture resembled delirium tremens with the patient being overactive, overtalkative and suffering from hallucinations. It is generally agreed that tetraethyl lead combines more readily with nervous tissue than do other forms of lead, hence one is likely to get a sudden attack of lead encephalitis without the ordinary signs of chronic lead poisoning. Kehoe[3] reported in 1934 that tri-ethyl lead may be inhaled in the form of finely divided dust by workmen cleaning the interiors of gasoline tanks with lead absorption and acute intoxication following. He states that the tetraethyl lead which is left on the sides of the tank decomposes after long standing to form crystalline tri-ethyl compounds. Apparently the clinical picture from tri-ethyl lead is similar to that from tetraethyl lead, and on the basis of Kehoe's report and personal communication it would seem that our patient had developed a lead encephalitis due to inhalation of tri-ethyl lead.

The interpretation of the pulmonary condition does not seem easy. Since tri-ethyl lead is intensely irritating and the patient presumably inhaled sufficient to produce a lead encephalitis, it is possible that the lung condition was produced by tri-ethyl lead. Dr. Sosman suggests this from his interpretation of the x-ray plates. Dr. Kehoe in a personal communication doubts the likelihood of this occurring. The length of time that ensued between the inhalation of the tri-ethyl lead and the development of the acute pulmonary signs, namely about six weeks, would also seem to argue against it. On the other

hand, it has been agreed by everyone who has examined the patient and the x-ray films of the chest, that the picture is atypical and does not conform to the usual picture of any of the well-recognized lung conditions. Without attempting to argue the matter further we merely raise this question.

The patient was also seen by Dr. Joseph Aub and Dr. Alice Hamilton.

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DISCUSSION

DR. JOSEPH AUB: It is important to remember that the whole picture of lead poisoning from organic lead compounds is entirely different from poisonings by inorganic compounds. The peripheral manifestations seem to be largely lacking, and lead colic and wrist drop do not seem to occur, while stippling in the blood only appears long after the acute cerebral abnormalities produced by these organic compounds. Kehoe has shown that, after ten to fourteen days, the distribution of the lead from lead tetraethyl differs little from that of inorganic compounds, but Gettler was able to find lead tetraethyl in the brain in a patient who died of poisoning from it. In the patient under discussion tonight, considerable quantities of lead could be isolated from the urine thereby showing that he had absorbed toxic amounts of some lead compound. While I have heard of other cases with severe mental disturbance such as this patient showed, most of the patients I have seen before have had only mild delusions and this patient stands out as unique in my experience in the severity of his symptoms with recovery.

DR. ALICE HAMILTON: I do not think I can add anything to this case except to review the history of tetraethyl lead. I heard of it first in 1922, when an expert from the du Pont Company asked me what I knew about it. I said I knew nothing, and asked him what the symptoms were, as he had seen them. He described an acute cerebral form of plumbism which was not encephalopathy as we know it but resembled delirium tremens, and he said that they held the ethyl radicle responsible, not the lead. A little over a year later the situation became a matter of sensational publicity when some twenty-eight severe cases occurred in New Jersey in the du Pont Works, and in the Standard Oil Works, where the compound was produced. The newspapers took it up, the health services of several states threatened to forbid the use of ethyl gasoline, the story crossed the ocean, questions were asked in the British Parliament, and Switzerland forbade the importation of ethyl gasoline. It was not so much the danger to the makers and blenders that aroused this excitement, but the fear that the lead discharged from motor engines might be a danger not only in garage work but in the streets, for in the course of combustion tetraethyl lead is changed to the chloride or bromide (according to the "carrier" used), both of which are unusually toxic compounds and are discharged in the exhaust gas. These public protests moved the purveyors of ethyl gasoline to ask the Public Health Service for a scientific study of the possible danger to the public, and this was done. The Service attacked the problem in a very practical way. They examined four groups of men:

First, taxi drivers who had never used lead gasoline; secondly, taxi drivers who had used it for two years; thirdly, mechanics in garages where it had never been used; fourthly, mechanics in garages where it had been used for two years; and fifthly, a control group of storage battery workers who were exposed to a known lead hazard of unusual severity. The investigators found what they considered evidence of lead poisoning only in the control group, and evidence of lead absorption in the fourth group but not in any of the other three. Kehoe of Cincinnati and his colleagues had previously made a similar study of filling station attendants and garage mechanics and had come to the same conclusion. The Public Health Service then announced that there was no reason why ethyl gasoline should not be put on the market. Some time later a British Royal Commission examined these findings and confirmed them. The Public Health Service has ordered the labeling of all pumps delivering ethyl gasoline, and has arranged with the purveyors for an investigation of every case of supposed plumbism resulting from contact with it. Leake, of the Service, and Kehoe of Cincinnati, have followed up all these cases and in 1932 I was told by the former that ninety-four illnesses in seventy-two individuals had been investigated but in none was lead found to have played a part. The present case belongs in the category of the early cases and is for that reason an interesting rarity, for under present conditions one would look for such an acute poisoning only as the result of an unusual combination of circumstances.

That there is danger in the use of this compound is recognized and vigilance must not be relaxed.

ADJUSTMENTS DURING FOUR YEARS OF PATIENTS HANDICAPPED BY POLIOMYELITIS

BY ELLEN H. BARBOUR*

THIS follow-up study was made with the purpose of determining the physical improvement and the emotional adjustment, during a period of four years, of sixty children who were victims of infantile paralysis. In 1930, in an article published in this *Journal* (207 : 1195 [Dec. 29] 1932) the degree of paralysis of these children was described in social terminology. Briefly, they were classified under the following headings: educationally paralyzed, implying that the individual was paralyzed to such a degree as definitely to impede his educational progress; occupationally crippled, meaning that although the child could lead a quite normal childhood, there would be a great possibility of his paralysis prohibiting him from certain occupations in later life; and socially handicapped, a disfiguring paralysis which although it

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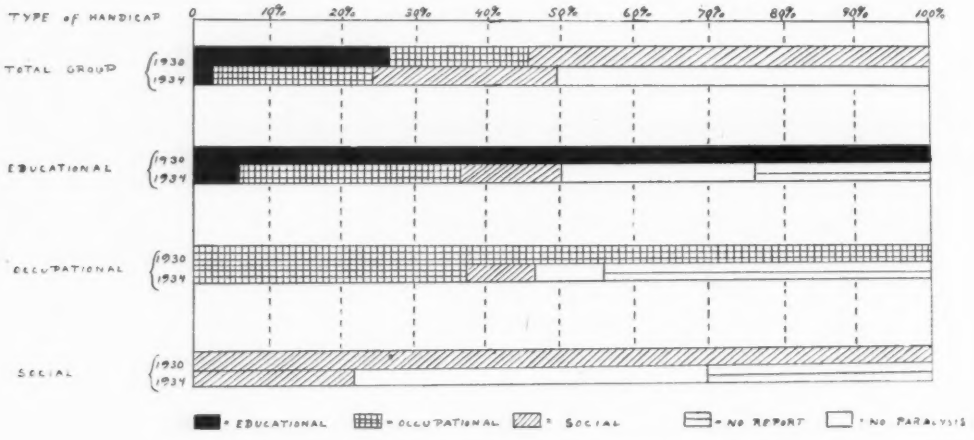
did not interfere with his physical life, might be a source of social embarrassment to him.

All the children, with but two exceptions, received approximately the same treatment respective to their individual needs, either in their homes or at hospital clinics. The accompanying chart shows the relative proportions of the three groups of handicaps in 1930 and their redistribution in 1934. In 1930 over one-half of all the children were in the social paralysis group; approximately one-fourth fell in the most severely crippled, educational group and a little less than one-fifth were described as occupationally handicapped. In the 1934 redistribution the paralysis of one-half of the total group had disappeared and one-half of those still crippled had only the mildest degree of paralysis. Slightly more than one in five were occupationally crippled and only one in fifty was severely limited in motion.

The charts also indicate the changes that have occurred during these four years in the groups. The educationally paralyzed seem to redistribute themselves either in the occupational or no-paralysis groups, with relatively few in the social class. The occupationally handicapped show the least change and the marked decrease of the social group would seem to indicate that the socially paralyzed child has an excellent chance of being completely normal at the end of four years of treatment.

The scholastic retardation was an interesting social factor of this study. Twenty-five out of forty children, at the end of four years, were not educationally retarded; nine were one year behind, four were two years and two were three years behind their classes. One of the two last mentioned attends the school for crippled children in Canton where she is rapidly making up her educational deficiency, and the other has undergone so many operations that, although between operations he is quite able to attend school, he has not yet succeeded in finishing one scholastic year since 1930. The twenty-one children who have completely recovered from the disease seem to have no darker memories of it than an attack of measles. They are a normally happy group, one of whom is described by his school-teacher as "the happiest boy in the class". Six out of the twenty children who are still paralyzed are emotionally maladjusted to their handicaps. The factors causing individual unhappiness may be more clearly discerned by the following brief case histories:

REDISTRIBUTION OF TOTAL GROUP OF PATIENTS HANDICAPPED BY INFANTILE PARALYSIS 1930 — 1934



REDISTRIBUTION OF TOTAL GROUP OF PATIENTS HANDICAPPED BY INFANTILE PARALYSIS 1930 — 1934

TYPE of HANDICAP	POLIOMYELITIS PATIENTS			
	CONDITION IN 1930		CONDITION IN 1934	
	NUMBER	PERCENT	NUMBER	PERCENT
TOTAL	60	100%	41	100%
EDUCATIONAL	16	26.7	1	2.4
OCCUPATIONAL	11	18.3	9	22.0
SOCIAL	33	55.0	10	24.4
NO PARALYSIS	0	0	21	51.2

CASE 1. B. P., a fourteen year old girl of good home environment, was left four years ago with a complete paralysis involving the facial nerves as well as those of her body. Today, the only traces left of the paralysis are a slight limp when fatigued and a twitching of the right side of the face when she becomes nervous or embarrassed. Nevertheless, she is extremely sensitive and bitterly resents any inference that she is not so strong as her sisters. As a result of this attitude she often overdoes to prove that she is normal. Whereas, formerly, she had a sunny disposition, she is now touchy and irritable due to a constant condition of fatigue. Her age coupled with her particular type of paralysis are probably the two most important factors in her emotional maladjustment.

CASE 2. M. P., now thirteen years old, was left with a paralysis of the right forearm and wrist. Her condition has not greatly changed during the last four years. Her arm is still useless and is noticeably smaller than the left. Any occasion, such as meeting people, which calls for shaking hands discloses her infirmity and causes her great embarrassment. She retires rather than face that painful ordeal. As in case 1, probably the two paramount causes of her unhappiness are her age and her particular type of paralysis.

CASE 3. Both legs of R. S., an eight year old boy, were completely paralyzed by the disease. His parents displayed such ignorance and stubbornness in refusing to permit the operation advised by the hospital and in ignoring the offer of suitable braces by the social service department, that action was threatened by the Society for the Prevention of Cruelty to Children. As a result of these controversies, the boy is completely unadjusted emotionally and physically to his handicap. He is very sensitive, cries easily and refuses to play with other children. The smallness of his brace undoubtedly causes him some physical discomfort. The parents' attitude of ignorance and stubbornness is surely the most important reason for his unhappiness.

CASE 4. M. K., a married man and the father of two small children, suffered from a weakness of the right shoulder and leg. During the past four years there has been but little improvement. Because of his physical disability he has been unable to retain such employment as he could find during this period. At the time of the 1934 visit, this family was receiving welfare aid and the patient's state of mind could only be described as desperate. Economic insecurity as a result of infantile paralysis is the direct cause for this individual's unhappiness.

CASE 5. P. B. was badly crippled when only two and one-half years old. Both legs were left quite useless. Now he walks with great difficulty in braces. His mother never permits him out alone since his balance is so precarious. She hesitates to take him among people because he is very sensitive and shrinks from even the stranger's casual glance. This little boy's hypersensitiveness would seem to be the main cause of his unhappiness.

CASE 6. Five year old E. J., an only child of adoring parents, suffered a paralysis of the right shoulder and arm. His condition has improved but little during the last four years. His parents took it so tragically and treated him as such an object for pity that some of their attitude was transmitted to the child, until he now feels himself to be an irrevocably damaged person. His physical disability has grown to such proportions in his parents' minds, and hence in his own, that he can think of little else. His fear of this "terrible thing" which has happened to him is pathetic. Obviously, an only child, devoted parents and poor psychology are the determining factors in this child's emotional maladjustment.

A METHOD OF REPAIR OF FEMORAL HERNIAE BY A FASCIAL STRIP FROM THE APONEUROSIS OF THE EXTERNAL OBLIQUE MUSCLE

BY GEORGE A. MARKS, M.D.*

IT has been said that at least one hundred methods of repair of femoral herniae are recorded in medical literature. One hesitates to add another method to the list. The procedure to be described below is offered, therefore, as a slight modification on a method already existing in many variations.

It is true that many femoral herniae are repaired successfully by simple ligation of the sac without attempt to obliterate the femoral opening. Recurrences are reported, however, in series by Watson, Coley and Erdman to vary from 4.85 per cent to 17 per cent.

Strangulation is dangerous in this type of hernia because of the small diameter of the ring. The Richter's type of strangulation in which only a portion of the wall of the intestine is involved seems especially apt to occur in femoral herniae.

The femoral opening by virtue of its anatomical construction lends itself with difficulty to obliteration. It is bounded anteriorly by the inelastic ligament of Poupart, medially by the fixed knife-like edge of Gimbernat's ligament and posteriorly by the pectineus fascia and muscle. On its lateral aspect lies only the femoral vein. None of these structures may be displaced, in the average case, with the necessary ease to insure permanent approximation, nor are they capable of exerting the sphincteric action found in the structures surrounding the inguinal rings.

Prior to 1890 ligation of the hernial sac alone was done. In 1892 Berger, and in 1894 Bassini, added the important feature of closure of the femoral canal and ring. They sought to accomplish this by suture of Poupart's ligament to the pectineal fascia (Cooper's ligament) and suture of the falciform process to the pubic portion of the fascia lata. Later, Coley em-

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ployed the purse-string suture of kangaroo tendon or chromic catgut to approximate these structures. Blake modified this by using a mattress suture for the same purpose, while Cushing placed two purse-string sutures in the same location.

Annandale, in 1876, had described the approach which splits the aponeurosis of the external oblique muscle, but this was not used extensively until Ruggi (1892), Lotheissen (1898), Gordon (1900) and Moschcowitz (1905) brought about various modifications.

Nelson's System of Surgery lists a number of curious procedures for the repair of this hernia, among them, the tacking of Poupart's ligament to the pubic bone by a metal staple and the use of heteroplastic flaps of fascia, periosteum, bone or muscle.

Two requirements must be satisfied to prevent the recurrence of a hernia repaired surgically; the defect must be obliterated by firm, relatively inelastic structures displaced from their original anatomical alignment as little as possible; these structures must be held in position by methods which interrupt as slightly as possible their viability and function. The principles of asepsis and hemostasis, of course, must be obeyed. In the methods described before, those which entail the approximation of Poupart's or Gimbernat's ligament to the pectineus muscle and fascia, attempt to displace an inelastic, firmly fixed ligament and anchor it to a structure which has very little strength. The resulting tension should defeat the purpose. If laxness in Poupart's ligament allows easy approximation to the pectineal fascia, this usually means a ligament of little durability.

Galli's technique of repair of herniae by the use of autogenous fascia removed from the thigh is widely recognized. McArthur's modification for inguinal herniae employs a strip of fascia from the aponeurosis of the external oblique muscle, left attached at its insertion in the rectus sheath.

Modification of this latter technique suggested itself as suitable for use in the repair of femoral herniae. By the modification described below, a sufficiently long fascial suture is available for obliteration of the femoral space without extending the operation to include removal of fascia from the thigh. While the latter maneuver is not a risk in younger patients, its avoidance is desirable in the older patients among whom the incidence of femoral herniae is high.

Technique: An oblique incision through the skin is made parallel to Poupart's ligament, and carried down to the fascia of the external oblique muscle. The position of this incision allows inspection of both sides of Poupart's ligament and exposure of the femoral opening from below if it is found necessary. Small hernial sacs may be pulled up from inside if not incarcerated.

The fascia of the external oblique is then split in the direction of its fibres. The contents of the inguinal canal are gently separated from the medial aspect of the ligament of Poupart until the femoral ring and the hernial sac are exposed. Further gentle blunt dissection around the ring is usually enough to allow the sac and its contents to be drawn in. If this is difficult or entails unwarranted manipulation, pressure on the sac from outside may be applied. In the event that this does not reduce the hernia, a small incision through the medial aspect of the ring in the direction of Gimbernat's ligament may be made.

After reduction, the sac is opened, the excess excised and the peritoneum closed by a purse-string, or running suture, reinforced by one or two mattress sutures.

A ribbon, five mm. in width, is then split from the upper leaf of the divided external oblique fascia, leaving it attached at its insertion in the rectus sheath above the spine of the pubis. Its outer end is cut across and fixed to a fascial suture needle with silk.

The fascial suture thus constructed is then threaded along the inner aspect of Poupart's ligament until the medial arc of the femoral ring is reached. From this point of departure, the suture is passed across the opening two or three times, depending on its diameter, anchored below each time by a bite which includes pectineus fascia, muscle and the underlying investment of the ramus of the pubis. At each crossing of the opening, the needle should transfix the strand just laid, approximating the strands and tending to obliterate small crevices. The terminal portion of the strip is usually long enough to allow a final interweaving in a horizontal or oblique direction. The end of the strip is anchored firmly at any point of the ring formed by Poupart's ligament by silk sutures. Any of the interstices left in the weave at this time should be closed by interrupted silk sutures.

The split in the external oblique fascia is closed by interrupted imbricating stitches, the superficial fascia and subcutaneous layer approximated by a fine running stitch of plain No. 00 catgut and the skin edges plicated with silk.

Three cases, one bilateral, have been repaired by the writer and two others by another at his suggestion, using the technique described above. The small number of cases, and the brevity of the postoperative interval, prevent conclusions being drawn.

Summary: A technique for repair of femoral herniae by the use of a strip of autogenous fascia from the external oblique aponeurosis, left anchored at its inner aspect, is described.

NEW HAMPSHIRE MEDICAL SOCIETY

CONGENITAL PYLORIC OBSTRUCTION*

BY CHESTER F. MC GILL, M.D.†

THE earliest reference to congenital pyloric obstruction was by George Armstrong in England in 1777. The case reported was the third similar one in the same family. All three died.

Again, Hezekiah Beardsley of Connecticut, in 1787, described in most interesting detail the clinical symptoms and postmortem findings in a five-year old with symptoms dating from infancy. According to Garrison's History of Medicine, Beardsley's account appeared in the earliest volume of medical transactions issued in this country entitled "Cases and Observations by the Medical Society of New Haven County in the State of Connecticut." Dr. Beardsley writes, "A child of Mr. Joel Grannis, a respectable farmer in the town of Southington, in the first week of its infancy was attacked with a pukeing, or ejection of the milk, and of every other substance it received into its stomach, almost instantaneously and very little changed. The faeces were in small quantity and of an ash color which continued with little variation until its death. For these complaints a physician was consulted who treated it as a common case arising from acidity in the prima via, the testaceous powder and other absorbents and correctors of acid acrimony were used for a long time without any apparent benefit. The child, notwithstanding it, continued to eject whatever was received into the stomach, yet seemed otherwise pretty well and increased in stature nearly in the same proportion as is common to that state of infancy, but more lean with a pale countenance and a loose and wrinkled skin like that of old people. This, as nearly as I can recollect at this distance of time, was his appearance and situation when I was first called to attend him; he was now about two years old. I was at first inclined to attribute the disorder to a deficiency of the bile and gastric juices, so necessary to digestion and chylification, joined with a morbid relaxation of the stomach, the action of which seemed wholly owing to the weight and pressure of its contents, as aliment taken in small quantities would often remain in it, till, by the addition of fresh quantities, the whole or nearly all, was ejected, but his thirst or some other cause, most commonly occasioned by swallowing such large draughts as to cause an immediate ejection, and oftentimes before the cup was taken from the mouth. It did not appear that he was attended with nausea or sickness at his stomach, but he often complained that he was choked and of his own accord would introduce his finger or the Probang so as to excite the heaving of the stomach and an ejection of its contents; the use of this instrument was generally necessary if the stomach did not of itself, in a few moments, discharge its contents. The choking would in that space of time become almost intolerable which by this discharge was almost entirely removed. In this situation, with very little variation of symptoms, he continued until death closed the painful and melancholy scene when he was about five years of age. He was uncommonly cheerful and active considering his situation. A number of the most respectable medical characters were consulted, and a variety of medicines were used to little or no effect. His death, though long expected, was sudden, which I didn't know until the second day after it took place. This late period, the almost intolerable stench, and the impatience of the people who had collected for the funeral, prevented so thorough an examination of the body as might otherwise have been made.

"On opening the thorax, the esophagus was found greatly distended beyond its usual dimensions in such young subjects; from one end to the other of this tube, between the circular fibers which compose the middle coat, were small vesicles some of which contained a tablespoonful of a thin fluid like water, and seemed capable of holding much more. I next examined the stomach which was unusually large, the coats were about the thickness of a hog's bladder when fresh and distended with air. It contained about a wine pint of fluid exactly resembling that found in the vesicles before mentioned and which I supposed to have been received just before his death. The pylorus was invested with a hard compact substance or scirrhosity which so completely obstructed the passage into the duodenum as to admit with the greatest difficulty the finest fluid; whether this was the original disorder, or only a consequence, may perhaps be a question. In justice to myself, I ought to mention that I had pronounced a scirrhosity in that part for months before the child's death.

"On removing the integuments of the abdomen, I was struck with the appearance of the vesica fellea, which was nearly five inches in length and more than one in diameter; it lay transversely across the abdomen, and was bedded into the small intestines, which were sphacelated wherever they came in contact with it; its con-

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 7, 1935.

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tents were rather solid than fluid and resembled flesh in a highly putrid state; its color was that of a very dark green, like the juice of a night-shade berry, and a fluid of the same color exuded through its sphacelated coats. The necessity there was of interring the body that evening, put a stop to any further examination.

"I should have been happy, gentlemen, if I had been able to have given you a more particular and accurate description of this very singular case, but the above-mentioned circumstances forbade."

About a half century later, Williamson of London, in 1841 and Dawosky in Germany in the year following, mentioned the projectile character of the vomiting and each reported a case with autopsy findings. In 1888, Hirschsprung awakened the modern interest in pyloric obstruction with his discussion of the clinical symptoms and presentation of two necropsy specimens before the German Pediatric Society. Following this, Ibrahim collected over four hundred cases about twenty years later. In 1896, Finklestein first called attention to the palpable tumor. The first surgical procedure was performed by Cordua in 1893 and was a jejunostomy. In 1898, Willyer of New York and Stern in Germany each reported a case of gastroenterostomy and this operation continued to be done for a number of years. In 1907, Fredet performed the submucous pyloroplasty by means of a linear incision down to the mucosa and suturing the longitudinal wound into a transverse one. Rammstedt, in Germany, in 1912, advised omitting the transverse suturing and leaving the wound gaping.

As to statistical data on the incidence of the condition, I can only cite the report of Hertz published in 1916, in which he states that 2.7 per cent of all children under one year show the condition. Walls, comparing appendicitis with pyloric obstruction, observed in a clinic of five thousand cases a year, less than five cases of acute appendicitis and thirty of pyloric obstruction, over a five-year period. There have been eight cases in the Portsmouth Hospital in the past five years.

A condition so common needs only mention of its cardinal symptoms: projectile vomiting, loss of weight, visible gastric peristalsis, with or without a palpable tumor, since the underlying pathology considered responsible for the symptoms is spasm of the pylorus with or without an accompanying hypertrophy. Also, the symptom of tumor may be so difficult to elicit, that failure to identify it ought not to militate against the diagnosis. In our small series of seven cases, the tumor was distinctly felt only twice. W. R. Meeker, quoting Strauss, states that "he has felt it in only twenty-five per cent of his cases and attaches no significance to it". In his own six operative cases, it was definitely found in two. These babies so afflicted are always constipated and the x-ray if employed will show evidence of obstruction. At the Children's Hospital in Boston, roentgenology is rarely done because it is not considered necessary, and it is not desirable that the stomach be full of bismuth at the time of operation. These cases, by the way, are all treated surgically. Recently the observation of the cold extremities has been suggested in addition to the above symptomatology.

For years, a great source of argument was the question as to whether the hypertrophy preceded the spasm or the spasm caused the hypertrophy. The pathogenesis, discussed at considerable length in the literature, has, at least until quite recently, remained rather obscure. Numerous explanations, all unsatisfactory, have been given for the spasm theory. Among these are the following: that the spasm followed by hypertrophy took place in utero as the stomach tried to rid itself of swallowed amniotic fluid; hyperadrenalism in the mother; phimosis causing irritation and accounting for the condition occurring so much more frequently in males.

Thomson, in 1897, stated that pyloric stenosis is a functional disturbance of the nerves of the stomach and pylorus. Haas suggests a disturbance in the vegetative nervous system with hypertrophic pyloric stenosis, an advanced degree of pylorospasm, and the two the most marked features of a general hypertonicity. Stenosis of the pylorus has been produced experimentally in dogs by Elsesser and in rats by Hoelzel and DeCosta. The latter found that gastric retention occurred with low protein feeding. Autopsy findings: stomach large, pylorus markedly constricted, and when the digestive tract was filled with fixing fluid shortly after death, the pylorus acted as if it were spastic.

In various gastrointestinal studies, they had often injected formalin-Zenker solution into the intact stomach, either through the duodenum or the esophagus, and in rats that had been kept on diets that tended to produce gastric retention, the passage of the fixing fluid was often obstructed entirely at the pylorus, while in rats that had not been kept on such diets, the fluid passed the pylorus easily. A pyloric hypertrophy such as occurs in infants was, however, never seen.

The proponents of the tumor theory hold first, that there is a primary developmental hypertrophy of the pyloric ring and canal and that the reduction of the lumen of the canal by the thickened muscle causes the obstruction; and, secondly, that there is an incoördination between the pyloric sphincter and the muscle of the pylorus, and that failure of the sphincter to relax causes the resistance which leads to hypertrophy of the pylorus. Apropos of this, Alvarez says that there is no definite pyloric

ring of muscle, for the pyloric muscle is but the lower end of the thick layer surrounding the pars pylorica of the stomach.

According to Sauer, the persistence of hypertrophy months after spontaneous recovery and in a cited case of a man operated for ulcer after a gastroenterostomy for stenosis, and in whom the thickened pylorus was seen to be still present, proves that the hypertrophy is not dependent on the spasm. On the other hand, that the clinical picture is dependent on the spasm whether accompanied by hypertrophy or not, seems to be borne out by the behavior of some of our cases, one of whom in particular did not vomit at all for four days and was discharged from the hospital only to return some days later vomiting as on his previous admission. Another, on whom a Rammstedt operation was done, began to vomit three weeks after the surgical treatment, which I am certain was complete, and showed visible gastric peristaltic waves with the vomiting of the projectile type continuing for about two weeks.

Many writers believe that the spasm involves not only the pyloric ring but the entire pyloric end of the stomach and even the whole of the stomach. Though no studies leading to the foregoing beliefs had included the nerves controlling this portion of the gastrointestinal tract, Moore quotes Elterich as stating that "the majority of writers attribute the disorder to an imbalance of the autonomic or involuntary nervous system, which of course consists of two divisions, the parasympathetics and the sympathetics, the functions of which are opposing.

Here, the physiopathologic views of Eric Prichard of London are of interest. He says, "The pyloric sphincter does not obstruct the outflow of the stomach contents owing to organic stenosis—it stops it because the opening is actually occluded or shut and refuses to relax. The normal resting condition of all sphincters is one of tonus, in other words, they are closed. The whole question of the innervation of the sphincter mechanism is very obscure, but it is agreed on all sides that when the sphincter is at rest it is closed and when it is in its active phase, it is open. But the exact manner in which the nervous system, or the pneumogastric nerve, controls the action of opening, is still in doubt. In the case of the pylorus, it appears that the peristaltic wave, controlled and initiated by the vagus, passes along the pyloric end of the stomach and finally terminates in relaxation of the sphincter proper. It commences in the muscles of the antrum and ends in an active phase of relaxation of the pyloric sphincter. The latter is a comparatively small structure, constituting an insignificant part of what is commonly known as the pyloric tumor in cases of hypertrophic pyloric stenosis."

With regard to the pyloric physiology, Gianturco of the Mayo Clinic reported in 1933 some results following experimental studies on the pyloric mechanism in which he had outlined the two curvatures of the stomach, the pyloric ring and the contours of the duodenum, with lead shot placed under the visceral serosa. Then with roentgenograms he took records of every phase of gastric activity. He found that food leaves the stomach when the pylorus and the duodenum are relaxed at the same time, and that relaxation of the pylorus alone is not followed by the passage of food. Alvarez states that the pylorus is opened by a wave of relaxation that normally follows a wave of contraction, and material is pushed out by the next wave of contraction which follows at a distance of several centimeters. Food goes into the pylorus which is open much of the time but passes through only at intervals. Forces below evidently balance those above. The duodenum, if not stimulated and caused to contract actively, will allow the stomach to empty.

As previously suggested, no cause for "the disturbance of the physiological function of the vegetative nervous system" had been developed, so it remained for Moore, Plymate, Brodie, Dennis and Hope, working in the Nutritional Research Laboratories of the University of Oregon Medical School upon problems connected with the effect on the young of inadequate maternal diets during pregnancy and lactation, to make some remarkable observations and to produce the disturbance experimentally in albino rats. Their research work concerned the effects of the lack of the vitamin B complex. Laboratory animals on diets containing two per cent yeast produced ten cases of pyloric obstruction together with hemorrhage of both the young and the mothers, and a high percentage of deaths with terminal paralysis. In percentage, obstruction occurred in 1.2 per cent of the young of the first generation, and in twenty-two per cent of the young of the second generation. Changing the method to the giving of .2 of a gram of yeast daily, excessive hemorrhage stopped and no cases of pyloric stenosis appeared. Brodie, reporting on the above-mentioned cases, states that at autopsy the stomach was distended to at least three times its normal size by tightly packed curd. The pylorus was tightly constricted but patent to the extent that a small probe could be passed with difficulty. In some cases, the pylorus appeared grossly hypertrophic. In the cases in which the condition was mild or was cured by atropine, polyneuritis was later superimposed upon the original condition. The pyloric obstruction is therefore considered as one manifestation of vitamin B deficiency with a resulting defective myelinogeny.

As to this last point, C. O. Prickett following his studies on the effect of a deficiency of vitamin B upon the central and peripheral nervous systems of the rat, thinks that the conclusions of these and other investigators as to myelin degeneration of the peripheral nerves may be in error due to the inadequacy of their methods

of preparation (Marchi). He found, however, definite changes in the central nervous system.

It has been suggested that the extent to which the findings on rats are transferable to man is something of a question. However, rats are omnivorous like man; both species develop lesions with inadequate diets.

The familial incidence of this condition though unexplained is nevertheless interesting. As previously mentioned, the case reported by Armstrong in 1777 was the third in the same family. Sauer speaks of two families in each of which there were three cases and twin boys in whom the disease was shown at autopsy. Finkelstein described four cases in one family and three cases in each of two other families. Caulfield noted two cases in successive generations. Roche reports the case of a boy, the sixth birth in a family, in which the fifth, also a boy, had pyloric stenosis.

Koplik ascribed the condition to a neurosis and in some cases, heredity. He had two cases in sisters whose husbands were brothers and sufferers from gastric neuroses. Both of these sisters had babies with pyloric spasm and stenosis.

With their experimental work in mind, Moore et al. ask whether or not this phase of the condition may be dependent upon the fact that food habits run through several generations. They also suggest the possibility that "a varying demand for the antineuritic vitamin may be the hereditary factor".

Ernberg in Stockholm suggests a hypothesis based on clinical observation. He noted the occasional admixture of blood in the vomitus observed in chocolate-brown streaks or the whole material of this color. There never was fresh blood or violent vomiting. The first symptoms set in suddenly—explained by a sudden irritation of the pylorus. He says that the rapidity of increase in the quantity of breast milk taken during the third to the sixth week makes demands on the unaccustomed stomach and pylorus and a small rupture of the pyloric mucous membrane is his conjecture. He has been unable to prove it histologically.

The trouble develops in males much more frequently than in females, the ratio being three or four to one. In seven of our cases, five were males and two females.

This is also said to be true in beriberi due to vitamin B deficiency and in polyneuritis occurring in animals and due to the same cause.

This mass of experimental evidence, though perplexing, forces the conclusion that any modern conception of the probable cause of pyloric stenosis, whether one believes it to be vitamin B deficiency or not, precludes the idea that all cases are surgical from the beginning.

T. H. Lanman and Patrick J. Mahoney in February, 1933 published a report of four hundred and twenty-five cases operated by the Fredet-Rammstedt pyloromyotomy at the Children's Hospital in Boston where it is the treatment of choice. The mortality was 6.3 per cent with 10.4 per cent in the early cases, reduced to two per cent in the later group. Since this report, however, they have done considerably more than one hundred cases with no mortality. In this connection, Haas says: "The reason the Rammstedt operation is superior to any other operation that has been attempted is that it does the same thing as medical treatment by severing the nerves through which the reflexes are produced. If the nerves are not severed, the results are incomplete after resection of the tumor."

That atropine affects the termination of the nerves and not muscles, accords well with the assumption that the spasm is produced as a reflex.

The usual treatment in this country has been atropine and thick cereal feeding and refeeding. Recently phenobarbital has replaced atropine to some extent. H. L. Moore says, "I believe the effects of this drug prove that the condition is also cerebral in origin and the phenobarbital inhibits emetic impulses carried by the gastric nerves to the vomiting center." Moore's cases average a week to ten days in the hospital, a shorter time than with surgery.

In addition, these babies must be given salt solution twice daily by hypodermoclysis and this must be done regardless of the control of vomiting.

J. B. Sidbury in 1927 described the good results in severe malnutrition, of blood transfusion in infancy. Blood increases food tolerance and resistance to infection. It has a food value and a vitamin content. It would seem, therefore, to be a most valuable addition to the usual treatment of these infants. It is obtained from the father—typing is not necessary—and it is given subcutaneously on the sides of the abdomen in amounts of 25 to 50 cc. per kilogram of body weight.

The addition of vitamin B to the diet gave no striking results over those reported similarly treated by others but without the addition of vitamin B. However, in the presence of cold extremities, subnormal temperature and poor appetite, it is invaluable.

No one of the mothers to whom we have given brewer's yeast in the past five years has had a baby who has had either pyloric stenosis or hemorrhage of the newborn.

In conclusion, I believe that with the present status of our knowledge concerning the problem, medical management should first be tried with the weight curve, determination of the amount of fecal matter in each twenty-four hours, the general condition of the patient, as stated by Sauer, the criteria for determining how long the treatment should be continued.

DISCUSSION

PRESIDENT LORD: I will ask Dr. Benjamin P. Burpee of Manchester to open the discussion on Dr. McGill's paper on "Congenital Pyloric Obstruction."

DR. BURPEE: Dr. McGill is to be congratulated on this paper, for again bringing to our attention the fact that this is a condition that does occur not infrequently in early infancy, and for stressing the fact that it is important that as early a diagnosis as possible should be made when the condition does arise, inasmuch as the treatment is always and completely surgical.

I believe that the history and symptoms of this condition are usually pretty clean cut and definite and that none of us will have much difficulty in the diagnosis if we will but remember that a sudden onset of vomiting at about the fourth week calls, not for a tinkering with the infant's feeding but for a careful examination of the upper abdomen while the infant is taking its food to determine whether peristaltic waves are present.

I believe that the important points in diagnosis are three in number. First, is the history. The onset is usually sudden. The mother can usually fix this onset almost to the hour that it began. This is between the third and fifth weeks. Vomitings beginning at birth are unlikely to be tumors, although I have seen one case in which this did occur.

Secondly, is the magnitude of the waves. In differentiating pyloric tumor and gastrospasm or pylorospasm, I believe that when the waves are very definite and high, often with more than one visible at a time, this is in favor of tumor. Peristaltic waves do occur in spasm but they are not so clean cut and definite.

The third point in diagnosis in differentiating tumor and spasm in doubtful cases is in the feeding of a thick cereal formula. If the infant persists in vomiting when this is fed the chances are overwhelmingly in favor of a tumor being present. Cases of spasm do not vomit thick cereal to any appreciable extent.

It has been my experience that feeling the tumor is difficult. As an interne on a service on which there was always at least one pyloric stenosis this did not seem so difficult. Of late I have not found it easy to feel the tumor. It is undoubtedly difficult, due to the location, but probably the answer is that it requires considerable time and patience in palpation.

Concerning the use of the x-ray in diagnosis this has always seemed to me to be an unnecessary procedure. It tells us nothing more than that we have stasis, which we already knew.

In the after-care preference is decidedly for whey and glucose. This provides an easily assimilated, non-curd forming food. Reporting sixteen cases operated by Dr. David W. Parker, it is of interest that the last six cases, fed on whey and glucose, with no mortality have shown an average gain of eleven ounces over a ten-day postoperative period. The previous ten cases, fed on various formulae, mostly low fat mixtures and lactic acid milk had a mortality of thirty per cent and over a corresponding ten-day postoperative period those that lived showed an average gain of only five ounces.

PRESIDENT LORD: I will ask Dr. E. L. Levine of Portsmouth to continue the discussion on this paper.

DR. E. L. LEVINE: *Mr. President and Gentlemen of the New Hampshire Medical Society*—I was greatly impressed with the report of the work done at the Children's Hospital, particularly the mortality rate, which is very commendable.

However, a report which was made in 1934 in the *British Medical Journal*, by Dr. Wallace of the Edinburgh Clinic in Scotland, is very illuminating to the extent that it goes to prove that surgery is not always the method of choice over medical treatment. Their mortality rate in that clinic is reported to be 24.8 per cent, which is practically twenty-five per cent. In other words, one out of every four patients died of some complication or another.

Perhaps outside of Boston and in other clinics, the mortality rates are much higher than reported at the Children's Hospital. This is very interesting because the pediatrician and the profession at large always think of the surgical treatment and do not attempt medical relief, consistent with the safety of the patient.

PRESIDENT LORD: Discussion is now open to guests and members of the Society.

DR. DAVID W. PARKER: *Mr. President, Ladies and Gentlemen*—I think that this group of men should be congratulated for their exposition of this very important and timely subject. There is no question in my mind but that this condition occurs very much more frequently than is recognized, and that many of these cases are carried along as feeding cases until the disease or condition is far advanced, and consequently the mortality raised.

This may be a bold statement, and I don't want you to take it in the way of criticism, but I don't think there is any particular reason why these cases should be missed for any considerable length of time.

As has been brought out, the symptomatology, the history, etc., is quite striking and quite definite. It isn't necessary to diagnose a tumor in order to make a definite diagnosis. If I have peristaltic waves, vomiting which has come on suddenly, more or less, and in a previously apparently healthy newborn, and the ejection of thick cereal gruel, with loss of weight, etc., I think it is very strongly diagnostic. My feeling is that very little time should be wasted on these cases in fooling around with changing foods and trying to get them as feeding cases. If they eject thick cereals for any considerable time, for a period of days, I am firmly convinced that we have a tumor at the pylorus and it should be investigated.

X-rays, as Dr. Burpee said, are absolute nonsense; they tell you nothing and are unnecessary.

It was rather interesting that Dr. McGill spoke of the familial instance. We had three cases in one family.

Now, the operation of choice is the Rammstedt operation, without question. From time to time, there have been articles published on modifications of the Rammstedt operation, frills, like putting the omentum up over the raw surface, making transverse cut at one end of your incision, that, to obviate the danger of getting into the duodenum. Incidentally, I might state that there is a distinct danger of that, because this tumor mass runs off very gradually into almost nothing, as you might say, into the mucous membrane of the duodenum, and in spreading your incision into the pylorus, great care must be exercised not to open the duodenum. It can be repaired, but it is not nice. It is a nuisance.

Now, the Rammstedt should be done very quickly, and there, I think, is one of the important things. These children are babies, are dehydrated and are poor surgical risks, and it is necessary to get in and get out just as quickly as possible, consistent with good work, so the less technical procedure that is carried out, the better. A small incision in the right upper quadrant over the left of the liver, identify the tumor masses with your finger, lift it into

the wound, incise, spread it with the forceps. Close it, and do it in five minutes.

There is another thing. These operations, although possibly they are emergency in some cases, should be treated as actual emergencies; that is, they should be operated on within five minutes of coming into the hospital. These children are dehydrated, almost mummified in the late cases, and if you are going to keep your mortality rate down, it is absolutely necessary to get fluid in. Fill them up with salt solution before putting them onto the table. I think that is one of the most important things connected with the operation. Don't forget that.

On transfusions, we have had no experience with them at all at the Balch, and so I don't think we would have saved any of our babies on that score.

Another important thing is the after-care. Dr. Burpee has given you statistics about the last sixteen cases fed on whey and carbohydrates; they used to say that the salvation of these children lay in the feeding of breast milk, and I think that is true, but I don't think it is so true as it was before we got on to this whey feeding.

DR. WINOGRAD: *Mr. President*—I wish to congratulate the speakers on their very complete and excellent papers, especially Dr. Burpee, for bringing out so clearly the difference between the two types of cases. Pylorospasm might be called a local picture of a general syndrome, the hypertonic baby. There are various degrees of hypertonicity. When we have a very hypertonic baby, we begin to wonder whether we have a pyloric stenosis. I feel, as they do at the Children's Hospital, that one must feel the tumor before operating, for, after all, operations are not the methods of choice, but especially in children the method of last resort. When a child is brought in to the office, it is very easy to tell a hypertonic baby; one sees a wiry infant with spastic, crossed legs, the so-called colicky baby of a nervous mother. Here heredity comes in, for the mother, also, is a high-strung person in most cases.

In those cases, great care, medically, may do much. In a small town like Nashua, with about 30,000 population, in the past two years, there have been only two infants operated upon for pyloric stenosis. Those children, in contrast to the hypertonic baby, were not hypertonic at all. A definite tumor was felt in both cases. Of course, in many cases there may be a combination of both. All the hypertonic cases have responded either with atropine or thick cereal. I might say that with atropine one must be courageous and at the same time cautious in its use. Some babies seem to have overdosage with a half drop of a one thousandth solution. They will become red and show dilation of the pupils. Other infants may stand twice or three times the adult dose, and may respond to large doses. Then, if that doesn't work, the thick cereal feeding usually will.

Some cases may be very bad and may show, as Dr. Burpee brought out, not the real wave that we see in the pyloric stenosis, but the suggestion of waves. I remember the case of a baby four months old; the child weighed eight pounds at birth, and at four months, it weighed six pounds. The vomiting was projectile in character, but that infant did gain, with the use of atropine and milk, and is today a fine child.

I might say this, too, that the simple things in infant feeding are much more important sometimes than the gross things, and are often overlooked because of their simplicity. For example, many babies find it difficult to digest fat; there may be severe vomiting with a great deal of cream. Skimming the milk might do more, sometimes, than some of the more radical procedures.

PRESIDENT LORD: Is there any further discussion of this subject?

DR. LOUIS C. AGER: My ideas on the subject of pyloric obstruction in infants are undoubtedly influenced by the fact that my knowledge of the condition goes back to the days when a posterior gastroenterostomy was the customary operation. Naturally we waited a long time before undertaking a procedure which was followed by death in a large majority of cases.

We ran through the gamut of feeding theories and when the thick gruel feeding was adopted we got very remarkable results in a goodly proportion of cases. It was a troublesome and messy performance to shove the food down the infant's throat with a tongue depressor and we had difficulty in many instances in having the parents continue the method long enough for safety. Statistics proved pretty conclusively that breast-fed infants were fully as subject to the condition as bottle babies and it was generally believed too high fat in the breast milk was a causative factor, producing hyperacidity. Hyperacidity and cyclic vomiting were the bugbears of pediatricians in those days. Perhaps the vitamins have corrected all that.

The Rammstedt operation was of course a tremendous step in advance but we had learned in the meantime that a very large proportion of the cases could be successfully treated without operation. My belief that the primary condition is spasm and that the actual stenosis is a secondary hypertrophy due to spasm is merely strengthened by the statement made here today that the tumor usually shows up at about the fourth week. The very definite symptoms of obstruction almost invariably appear in the second week after birth. I agree absolutely with the previous speaker that, if the infant lives to the fourth week, operation is indicated, and I would go farther and say that in most cases surgical intervention should be made before that time.

DR. FRED B. LUND: We have just heard a little conservatism preached. I think the definition of conservatism is really the kind of conservatism that saves life, and not the kind that avoids a simple and life-saving operation.

After you have seen these cases, with a certain onset of symptoms in previously healthy children, if you wait until you can feel a tumor, you usually wait too long. Children change so quickly, so much more quickly than adults. They are growing fast, and they go off just as fast as they come on. The idea of waiting until you have tried every possible thing strikes me as the very best way to get a high mortality, and the statistics that have been shown here today demonstrate that to be bold, if you can call it being bold, is really being careful; therefore, an early operation is really being careful.

DR. FRANK N. ROGERS: There is a danger point, beyond which it is not advisable to go. Most doctors who are experienced with this condition will get this child into the hospital and operated upon as soon as possible.

An economic point is this. In large hospitals, there is a tendency to operate as soon as possible and send them home in about three weeks to avoid cross infections.

The cases that we have had that I have observed in the city have been cases which were so early that it was impossible to feel the tumor on most of them.

One case that Dr. Parker mentioned that I had to do with had three in one family; they were all breast fed, and the mother carried the baby around

under her arm and fed it almost constantly. When the baby entered the hospital, it would have made a gain, if all things had been equal. But the vomiting was persistent and characteristic of hypertrophic pyloric stenosis, and after going over the case thoroughly in the hospital and giving it sufficient time, the case was operated upon and a sizable tumor was found. The baby was discharged in three weeks, on the road to recovery. And that has been the story as time has gone on.

Men are more alert in making these diagnoses today and I think that we are getting into the hospitals better risks than we ever had before.

I think the objection to surgery in the past has been that the cases were such poor risks when they entered the hospitals that the surgeon was almost discouraged from the start.

DR. HERBERT L. TAYLOR: Cases of pyloric stenosis in children, I think render no difficulty in the matter of diagnosis, and the treatment, of course, should be early and it should be prompt.

Cases of pyloric stenosis, of course, present two kinds; they present a hypertrophic kind, in which the circulatory muscular fibres are greatly enlarged and increased in number, and you also have the form of pyloric stenosis in which it is simply a spasm of the pylorus. In that instance, it is supposed to be due to some defect of the vagus nerve in that region.

Of course, the treatment in the hypertrophic type is the Rammstedt operation, which can be done very quickly under novocain, and the results should be perfect in regard to the patient getting well, if the case is done early.

The type of pyloric stenosis due to spasm often presents intermittency in the vomiting. They will vomit for a while, and then the child will go along, apparently in perfect health again, and then start in vomiting. That is quite often the way with the type which is due to the spasm.

PRESIDENT LORD: If there is no further discussion, I will ask Dr. McGill to close the discussion on his paper.

DR. CHESTER F. MCGILL: The paper was not offered with the idea that vitamin B deficiency is necessarily the panacea. I daresay, however, that seventy-five per cent may, in the future, be treated successfully, by medical management. The other twenty-five per cent will require, some time or another, the Rammstedt surgical operation.

I hoped that this paper would be provocative of greater interest on the part of all members of the Society, particularly with a view to ascertaining, if possible, whatever information may turn out to be of value with regard to the mothers' diets. This, of course, concerns the obscure etiological factor, particularly with regard to vitamin B deficiency.

THE AMERICAN PUBLIC HEALTH ASSOCIATION CONVENTION

Leaders of public health and preventive medicine from every state of the union and from several foreign countries are to be at hand for the sixty-fourth annual convention of the American Public Health Association to be held in Milwaukee from October 7 to 10.

Workers in all fields of public health, medical men, nurses, specialists in research and others in allied activities are to be among the 3,000 delegates expected at the convention.

Important developments of the year in preventive medicine, in the extension of public health work and in the constant war on contagious disease will be unfolded at the gathering.

Simultaneous with the association's sessions, ten other closely related organizations are to convene. They are the Association of Dairy, Food and Drug Officials, American Association of School Physicians, Association of Women in Public Health, Conference of State Sanitary Engineers, Conference of State Laboratory Directors, Conference of Wisconsin Health Officers, State Registration Executives and State Directors of Public Health Nursing, International Society of Medical Officers of Health, and National Committee of Health Council Executives.

Over 400 papers are to be presented, dealing with such widely varied subjects as mental hygiene, diphtheria and scarlet fever immunization, poison in food, public sanitation, treatment of sewage and polluted waterways, public health engineering, planned milk control, laboratory studies with toxins and vaccines, tuberculosis, milk-borne disease, water purification, new methods in reporting vital statistics, industrial hygiene, food and nutrition.

Sessions of the association itself are to be divided among its ten constituent sections—health officers, laboratory, vital statistics, public health engineering, industrial hygiene, food and nutrition, child hygiene, public health education, public health nursing and epidemiology.

There will be authoritative presentations on amebic dysentery, hemolytic streptococci, whooping cough, gonococcal infection, tuberculosis and undulant fever. Papers are to be presented on filtrable viruses, and on prophylactic and therapeutic vaccination. Recent progress in biological products, and research accomplishments of the year in the milk and dairy field will be on the program.

Among the outstanding physicians and health leaders who will speak at the convention are Dr. E. L. Bishop, President of the Association and Director of Health of the Tennessee Valley Authority; Dr. Walter H. Brown, President-Elect of the Association and Professor of Hygiene at Stanford University in Palo Alto, Calif.; Dr. Herman N. Bundesen, President, Board of Health, Chicago; Dr. W. G. Campbell, Chief of the United States Food and Drug Administration, Washington, D. C.; Dr. A. J. Carlson, Professor of Physiology, University of Chicago; Dr. Haven Emerson, Professor of Public Health Practice, Columbia University; Dr. John A. Ferrell, Associate Director, International Health Division, Rockefeller Foundation, New York; Dr. Morris Fishbein, Editor, *Journal of the American Medical Association;* Dr. Grant Fleming, Professor of Public Health, McGill University, Montreal; Dr. Iago Galdston, Executive Secretary, Medical Information Bureau, New York; Dr. John P. Koehler, Milwaukee Health Commissioner; Dr. Carl S. Pederson, Chief in Research, Agricultural Experiment Station, Cornell University, and health commissioners of most large cities and research men on the staffs of foundations and major industries.

This congress is designed to appeal to members of the public health and medical professions alike. Headquarters are to be at the Milwaukee Auditorium. Scientific and educational exhibits will be staged. Entertainment and social events have also been planned.

MEDICAL PROGRESS

PROGRESS IN LARYNGOLOGY

BY LE ROY A. SCHALL, M.D.*

IONIZATION of the nasal mucous membrane has been given a new impetus by the work of Warwick and the enthusiastic attitude of the manufacturers of his electrical apparatus. Theoretically it is based upon the fact that the metallic solution placed in contact with the nasal mucous membrane, results in the precipitation of the proteins of the superficial cells. The method has been tried by Tobey[1], Alden[2], Hollender[3], MacFarlan[4] and others. Tobey frankly states that it has not been tried in a sufficient number of cases, nor has sufficient time elapsed to judge the relief adequately, which may be but temporary, given 85.6 per cent of his patients.

Whether the effect of ionization is anything more than a cauterization of the nasal mucous membrane is questionable. It remained for Palmer[5] to have the courage of his convictions and treat a group of thirty patients with vasomotor rhinitis by a local application of concentrated phenol. The immediate effect of this treatment was exactly that which was obtained by the ionization instrument of Warwick in that the mucous membrane showed a greyish, white discoloration followed by edema and obstruction with hypersecretion. Palmer had excellent coöperation from his patients, as they voluntarily permitted biopsies to be taken. The microscopic examination revealed that there was an increase in the connective tissue of the tunica propria with a diminution of the edema and vascularity. Of thirty cases treated by Palmer, twenty-four or 80 per cent showed definite improvement and twelve were free from symptoms for periods of from three to nine months.

McMahon[6] approaches the subject of ionization from the pathologic point of view, by determining its effect on the nasal mucous membrane of dogs. He found that ionization is followed by a desquamation of the superficial epithelial cells, by an increased fibrosis of the connective tissue and by multiple abscess formation deep in the stroma. There was also a polymorphonuclear leukocytosis and an extravasation of red blood cells free in the tunica propria.

Fenton[7] bluntly states that "ionization as such does not do anything more than damage the mucosal tissues of the sinuses".

The fact that hay fever is unknown in Japan, but that on American soil over three per cent of the Japanese population contract hay fever, led to Hara's[8] investigations. He found that the concentration of pollens in the air in Japan was much higher than that in Los Angeles, where he made his American studies. He also found all the Japanese pollens to be large, heavy and non-spiculated. He believes that the high humidity and abundant rainfall in Japan prevent dissemination of the light pollens which are usually associated as the causative factors of hay fever.

For many years mucous nasal polyps were described as myxomatous tumors. It is now generally conceded that they are merely exuberances of the nasal mucous membrane. Kern and Schenck[9] believe that allergy is a constant factor in the etiology of all nasal mucous polyps. In their experiences all patients with mucous polyps have been found to have a general history of allergy, a family history of allergy, or positive skin tests, and usually all three.

TONSIL

The tonsil problem has been revived. For many years a paper on the subject of tonsils would have no place on a scientific program. The subject was thought to have been exhausted. A few years ago Kaiser startled the pediatric world by a publication of a large set of statistics from the school children of Rochester. The result of his figures showed that in his locality, the operation of tonsillectomy, does not in all cases, accomplish what the medical and lay public believed it would. In other words, he found that deep respiratory infections, namely, laryngitis, bronchitis and pneumonia occur more frequently in tonsillectomized children.

In a recent paper, Kaiser[10] gives his findings following a reëxamination of these children, ten years after operation. His statistics again show that the instance of bronchitis and pneumonia has not been lessened by the removal of the tonsils. He states, however, "My studies and end-results of tonsillectomy indicate that some physicians have underestimated the importance of the tonsils as a factor in disease. The outstanding problem with the physician is to discover a more accurate way of diagnosing diseased tonsils. There is no known therapeutic procedure that is of greater value to a child than the removal of diseased tonsils. The failures are largely due to the wrong diagnosis."

In the same symposium, Nissen[11] gives the result of his work, based principally on a survey of patients of the Robert B. Brigham Hospital. He found that of the arthritics, only 10

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per cent had tonsillectomy performed before the onset of their arthritis. Of the 90 per cent that had retained their tonsils, with the onset of arthritis, only 45 per cent had tonsillectomy, after the onset. In other words, this statistical study, showing 55 per cent of a group of arthritics, having tonsils intact for many years after the beginning of their arthritis, should answer the accusation as to the wholesale removal of tonsils in arthritis. Nissen very clearly brings out the fact that there should be a relationship between the focal and systemic infection before operation is advised and that after an operation is decided upon, the proper time for this procedure is when, not only the tonsillitis, but the arthritis, is in the state of absolute quiescence. Under no circumstances should an operative procedure be undertaken if the patient's disease does not reach a period of quiescence with measurable improvement.

Mullin[12] also pleads for a very thorough study of the patient before deciding that a disease is due to focal infection. He says, "I can only reiterate the caution and advice not to operate at random on a patient with focal infection, and let the failure to obtain results tell you that his case needed more study. First, do everything else that might help and then, after time and careful study, resort to surgical removal, only if there are definite indications. I wish also to emphasize the fact that care must be taken in training specialists, so that they become doctors and diagnosticians first and surgeons afterwards; that they do not hold out a tonsil and adenoid operation as a panacea for all ills and perform this operation indiscriminately without due and weighty consideration."

This is exactly the same attitude taken by Coates[13] who very properly says, "It is usually possible for the painstaking laryngologist to determine the presence or probably absence of chronic sepsis of the tonsil. Where the tonsil is very definitely diseased, its removal for prophylactic reasons will often give striking results. When the tonsil is removed on mere suspicion, or after hasty and inadequate examination, the guess may as well be wrong as right. When the septic tonsil is discovered in association with some systemic disorder, the diagnosis of tonsillar infection as the cause of this disorder can only be satisfactorily arrived at after meticulous study and consultation on the part of the laryngologist with the attending pediatrician, internist or surgeon."

Newhart and his co-workers[14] have been studying the question of tonsillectomy in the tuberculous. Of 324 tonsillectomies in tuberculous patients, only one operative case, prior to 1926 showed an unfavorable course clinically. They believe that this high percentage of favorable results can be obtained only by a very careful selection of patients and that the operation must be guarded by a very careful technique and by following a very carefully planned program for after-care.

Schall[15] analyzes a group of 230 cases of cancer involving the tonsil. The outstanding fact is that the majority of these patients endured their symptoms for from three to six months before consulting a physician. As a result of this analysis, it may be said that surgery has no place in malignant disease of the tonsil. The most encouraging results are obtained by radium implantation in conjunction with external radiation.

THE COMMON COLD

The common cold has continued to be a subject of study. Most investigators believe that the mucous coat which lines the nose plays an important part in the prevention of infection. Lucas and Douglas[16] believe that the normal mucous coat is composed of two layers, an inner one of fluid, fluctuated by the cilia and an outer layer of mucus which carries away débris and foreign particles. The inner layer flows with the vibrating of the cilia against the under surface of the inner layer. Therefore, they believe that the rate of flow of mucus cannot be used as a reliable measure of the ciliary movement.

It is Proetz's[17] opinion that where this mucous sheet is broken by a change in the surface epithelium, that bacteria can penetrate into the mucous membrane at these areas.

Hilding[18] found that these changed areas of epithelium are sterile and it is his opinion that a powerful bacterial enzyme, which he has designated as "lysozyme", acts as a protective agent.

It is suggested by Negus[19] that it is the changes of pH which paralyze the cilia and not the action of bacterial toxemias.

Tweedie[20] has studied the pH reaction of the nasal secretion by a colorimetric method and his findings suggest that the mucus of the normal nose can control pathogenic bacteria. It is not until the reaction becomes disturbed that the pathologic bacteria can retain a footing. However, Hilding[18] found that pH was but little disturbed by pathologic conditions.

SINUSES

American laryngologists are quoted freely by Tilley[21] in his Semon Lecture on nasal sinuses. He accepts Schall's histopathologic classification of sinus disease but adds an additional type; one in which bone changes have taken place. In discussing the operative treatment of chronic maxillary sinusitis he advocates the Caldwell-Luc operation for all cases except for those of the simple edematous type.

In Goodyear's[22] experience, the intranasal operation, in which a large opening is made beneath the inferior turbinate and the antrum packed with iodoform gauze which is left for three to nine days, produced as satisfactory results as the external operation.

Stevenson[23] reports seven cases of infection of the maxillary sinus in which fungus forms were found in the pus recovered from these sinuses. These fungus forms closely resembled those of mucor histoides. Four of the cases classed as chronic sinusitis had recurrent infections, while under observation, and in two of these cases fungus forms were also found in infections recurring after some months. The findings indicated that fungi were present as active agents, rather than as saprophytes.

From an analysis of his postoperative results, Hill[24], theorizes as to the causes of failure in a certain percentage of sphenoid operations. Most of the older authorities considered the intranasal approach quite satisfactory. There is, however, a growing conviction that the external approach to the ethmoid-sphenoid is not only the safest, but that more thorough surgery can be done. Even with external surgery, subsequent contracture of the operative opening may occur following sphenoidectomy and nullify the effect of the operation, even after complete removal of the anterior wall, including the pars ethmoidalis.

PHARYNX

Kully[25] classifies cysts of the nasopharynx as: (1) cysts of the mucous glands, the true retention cyst; (2) retention cysts of the pharyngeal bursa, described by von Luschka, Thornwaldt and others; (3) degenerative cysts, representing vacuolization of the germ centers with subsequent cyst formation and (4) bronchiogenetic cysts, situated in the lateral wall of the nasopharynx beneath the ostium of the eustachian tube. The symptoms are postnasal discharge, posterior cervical adenitis, occipital headache, otalgia and unexplained fever. Kully pleads for a more careful and thorough examination of the postnasal space.

Mattick and Thibaudeau[26] found but nineteen plasma cell tumors of the upper air passages in the literature. They report their case of plasma cell tumor occurring simultaneously in the nasopharynx and hypopharynx. These tumors display characteristics ranging from inflammation to malignancy. Very few show lymphnode involvement of recurrence after removal.

Pollock[27] reports two cases of plasmacytoma from the Massachusetts Eye and Ear Infirmary. He emphasizes the fact that they must be differentiated from multiple myeloma which can be readily done by careful roentgen examination of the skeletal system.

LARYNX

Kernan and Schugt[28] discuss abscesses of the larynx. The symptoms are pain low in the throat, hoarseness and dyspnea. Local examination shows an obliteration and bulging of the pyriform sinus. The operation proposed and carried out on eight cases by these authors is an external exposure of the thyroid cartilage with a trephine through the cartilage to reach the abscess.

Both Wilkinson[29] and Tucker[30] report a case of congenital web of the larynx.

An isolated amyloid tumor of the larynx without any demonstrable underlying local disease or general amyloid disease was reported by Kramer and Som[31].

In the past year many papers have appeared on the treatment of cancer of the larynx. An increasing number of radiologists are reporting their results by Coutard's fractional method of treatment and laryngologists summarize their cases treated by surgery. All laryngologists plead for early diagnosis. The outstanding symptom in malignant disease of the larynx is hoarseness, yet this symptom is ignored for months and even years. All writers agree that biopsy does not produce metastasis if surgical removal takes place soon after the biopsy.

Hirsch and Baum[32] believe that in intrinsic laryngeal cancer, clinical cures can be obtained by roentgen ray treatment. In such cases surgery gives equally good results, but with a lesser degree of conservation of function and with an average operative mortality of about 15 per cent. In extrinsic cases, the prognosis is usually unfavorable, owing to the tendency to rapid spread and metastatic glandular involvement. In this group, surgery gives a high mortality and offers no assurance of cure. Roentgen therapy may produce a clinical cure in this group with cervical involvement, or if the case is hopelessly advanced, it may offer palliation, comfort and prolongation of life.

Clerf[33] in fifty-eight patients with cancer of the larynx treated by laryngofissure had five deaths from postoperative complications. Thirty-five of these patients, three years after operation, have been traced and are free from recurrence.

Jackson[34] reports on seventy-five patients treated by laryngofissure with 82 per cent alive and well for periods from five to twenty-one years.

Tucker[35] reviews 200 consecutive cases of cancer of the larynx. In fifty-eight of the cases laryngofissure was performed; in thirty-one, laryngectomy and in fifty-four cases x-ray and radium were used. Only three of these fifty-four cases are alive after three years.

Another review of cancer of the larynx is by Beck and Guttman[36] who summarize their results with 500 cases. Laryngofissure gave a cure in 80 per cent and laryngectomy in 59 per cent, at the end of five years.

Equen[37] also writes a very good paper on cancer of the larynx, reviewing the symptoms

and urging early examination of the larynx in each case of hoarseness.

The effect of radium on the laryngeal cartilages has been the subject of investigation by Arbuckle[38]. His study must be regarded as a preliminary report, as the amount of radium used was so small and the distance from the cartilage so great that proper evaluation of the effects of radium cannot be made.

Nelson and Hirsch[39] warn radiologists that they should safeguard their patients against radiation injuries, especially when sensitive tissues, such as the neck, are being treated. They present an autopsy report of radiation reaction with necrosis of laryngeal structures following radiation of the larynx.

In view of these surgical results laryngologists are loath to try radiation on operable cancer of the larynx. Radiologists are treating only those patients who refuse surgery; therefore a just comparison cannot be made until a sufficient number of cases have been treated and sufficient time has elapsed to evaluate the end results properly.

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THE MORTALITY RATE

According to precedent, the beginning and ending of each year give the highest mortality rates in the returns from eighty-six cities of the United States. These figures are fairly representative of averages throughout the country.

Beginning with January, 1935 the average rate in these cities has dropped from 13.2 per thousand of the population to 9.3 in the first week of September. This decline has been very close to that of the preceding year, except for a brief rise in the last week of July, 1934, when it reached 12.2 but subsided quickly to 9.3 the last of August, which is the same figure as that for the present year.

If the usual trend takes place, the mortality rate will now rise steadily through September and October with a more rapid increase up to January 1936.

It will be interesting to note whether the slightly higher rates of the years before the depression are to be repeated or whether there will be a lowering of mortality rates in the years to come.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21381

PRESENTATION OF CASE

A twenty-eight year old married American school-teacher entered complaining of exhaustion.

During the two years before admission she felt very tired upon completing her day's work. During the past five months her fatigue increased and a faint dark red mottling appeared on her face. Two weeks before entry a similar mottling appeared on her neck and legs and an ecchymotic spot about five centimeters in diameter on the right thigh. One week before entry she developed a sore throat and a cough which later persisted and was associated with grayish-green, occasionally blood-streaked sputum. She had some bleeding from her gums and a slight nosebleed. Her last period started nine days before entry and was marked by profuse flow and clots. She had very severe pain during the first day.

Her mother, father and three siblings were living and well. There was no history of bleeding. She had always lived in Massachusetts. She had the usual childhood diseases and a tonsillectomy and adenoidectomy fourteen years before admission.

The past history is non-contributory.

Physical examination showed a well-developed and nourished woman lying in bed in no acute distress. Her gums were bleeding and some blood was exuding from the left nostril. There were purpuric blotches over the legs and arms. Examination of the heart, lungs, and abdomen was negative.

The temperature was 100.8°, the pulse 108. The respirations were 20.

Examination of the urine showed a specific gravity of 1.012 and a very slight trace of albumin. The blood showed a red cell count of 2,260,000, with a hemoglobin of 56 per cent. The white cell count was 18,800. A smear showed the absence of platelets. No differential count is recorded. There was no clot retraction in forty-eight hours.

A splenectomy was performed soon after admission. She had an uneventful convalescence and was discharged approximately three weeks after admission. The last blood smear showed an increase in the number of platelets but was still within the limits of low normal.

CLINICAL DISCUSSION

DR. BETH VINCENT: Probably if they had taken the bleeding time on this patient it would have been delayed. I dare say it was done by Dr. Reynolds before she entered the hospital. There were one or two features of this case that were not so favorable as might be inferred from the history as given in this report. Before the patient came to the hospital she had more bleeding than is indicated in this history, because splenectomy was considered before she got to the hospital. Then the note on the physical examination says "a well-developed and nourished woman lying in bed in no acute distress." That hardly paints the picture. She came in at six o'clock. I was talking to the husband downstairs and was summoned to the eighth floor. She was lying on her side and blood was pouring out of the left nostril, so much so that while we planned to take the spleen out the next day we had to alter our procedure, pack the nostril, transfuse the patient and do the splenectomy that morning.

This is a case of thrombopenic purpura. It is reported that the mortality is higher if you take the spleen out during the attack of hemorrhage. That is true; but transfusion combined with splenectomy is the most effective procedure to control the bleeding. Therefore, the more acute the bleeding the more acute the necessity of operation in spite of the risk; but at the same time if you could be certain they would survive the bleeding you would be glad to postpone the operation until a quiescent period. It is like an appendix; if soft avoid the operation in a very acute attack. There is one factor in the blood picture that Dr. Reynolds can comment on that is a little unusual.

DR. GEORGE P. REYNOLDS: Certain things in the history are worth going over because the history as presented is not accurate. In the first place she had been flowing for twenty-one days when she was admitted instead of nine as the history states. Her menstrual history is worth going over in detail because it is significant, especially in view of later developments.

She started menstruating at thirteen, always had scanty periods, and had sometimes gone as long as four or five months without periods, particularly during trips to Europe or any changes in environment, but also she had often skipped periods at home. She never had a profuse period prior to the present illness. The last period before the present illness was distinctly scanty. The September period started twenty-one days before admission, and while it had never been really profuse as judged by normal standards, she flowed more each day than was customary for her.

For two months prior to operation she had noticed easy bruising, and had had frequent though not severe nosebleeds. Moreover, she

had had a little, constant oozing from her gums during this period which is of some significance since she had never had it before.

There are also certain other factors in the history that are worthy of our consideration. In the first place throughout life she had tended to be a very healthy person and was very active and rather high-strung. For two years prior to the present illness she had been working extremely hard and was rather tired every night, but in evaluating the amount she did this fatigue did not seem to be inconsistent with the degree of activity. During the last three or four months before admission, however, she had become abnormally fatigued, although doing less than usual. Throughout life she tended to be obese and periodically had gone on very extreme dietary restrictions. We do not know the cause of this disease, but the more I see of blood diseases, the more I wonder if semi-starvation over a period of weeks does not do something to the bone marrow. This individual had frequently lived for days, or even weeks, on nothing but orange juice or a similar dietary insufficiency. Moreover, her diet for the last two years was inadequate in protein, although all of her extreme dietary restriction was before that time. She had usually taken one helping of meat a day, but because she thought meat was fattening, and moreover did not like it, the amount was extremely small. That is, of course, significant in an anemia history, but I do not know if it is in this disease. There was no past history of abnormal blood loss.

When I first saw her two weeks before admission the red blood cell count was 3,800,000, the bleeding time was normal, and there was no clot retraction in forty-eight hours. The clotting time, however, was not very much prolonged. The platelet count was recorded as 260,000 but I think we are often fooled by platelet counts which are at best inaccurate. The smear that I saw had very few platelets, approximately one to each high power field, and I think that this is a more accurate estimation of her platelets than the count. As Dr. Vincent said, we do not like to operate on these cases during the acute stage. At that time her flowing seemed to be decreasing. The bleeding from the gums was minimal, and there were no nosebleeds. We therefore felt justified in trying other measures before suggesting operation. We first gave her a well-rounded diet with plenty of protein, including large amounts of whole liver. Occasionally even in fairly severe thrombopenic purpura whole liver has proved to be effectual, especially when there has been an antecedent history of dietary deficiency. In this case it had no effect.

The next thing we tried was transfusion. Looking back I think that possibly we might have anticipated the emergency a little earlier by the fact that transfusions did not have any appreciable effect. I think also we were swayed somewhat by the fact that there was very little bleeding other than menstrual, and we rather counted on the belief that menstrual bleeding in this disease is of less serious significance than other abnormal blood loss. She had four or five blood transfusions without any definite decrease in the menstrual flow.

She was then brought to the hospital, and as Dr. Vincent said, on admission it was obvious that the situation was quite serious and that an emergency operation was definitely indicated. Almost immediately after removal of the spleen all signs of bleeding stopped, not only the acute nasal hemorrhage and the oozing from the gums but also the menstrual flow. This is what usually happens but the interesting feature in this case is that it was twelve days after operation before she showed any increase in platelets. There was no bleeding, no purpura, but repeated blood smears showed only from one to three platelets per high power field. We do not know how to account for that. It is as far as I know a unique situation.

Just a word as to the prognosis which we felt justified in making at that time. Simply because she stopped bleeding her immediate prognosis was thought to be good. But at operation there was some evidence of cirrhosis in the liver which makes our prognosis a little more guarded than in the ordinary case.

It so happens that I saw her the day before yesterday when she came in because of slightly excessive menstrual flow. Her blood examination at that time was as follows: red blood cell count 4,800,000; hemoglobin 105 per cent by the Sahli method; histological appearance of the cells normal; platelets estimated as normal or increased; white cell count 11,400. The differential count showed only 30 per cent polymorphonuclear cells, which Dr. Minot feels is not a good prognostic sign. In all previous counts both before and after operation she had at least 70 per cent polymorphonuclears.

She looked extremely well and I felt that her prolonged bleeding with this period was not in any way related to the blood situation. She was seen yesterday by a gynecologist who reported that the flowing had practically ceased, but that examination revealed an extremely small, immature uterus. A pelvic examination had been made at the beginning of the present illness and was reported as normal. I think if we had realized that the uterus was immature we might have paid more attention to the flowing and felt that it was more abnormal than we did. In thrombopenic purpura one should regard any evidence of bleeding with a good deal of apprehension, and one always hopes to postpone operation until the acute phase is over. In this case we were less apprehensive than the subsequent course proves we should have been because she looked so well and because the bleed-

ing was largely menstrual rather than from other parts of the body. If we had known that the reproductive organs were so immature we might have regarded the uterine bleeding as distinctly abnormal and have been a little more concerned.

Preoperative Diagnosis

Thrombopenic purpura.

Pathologic Diagnosis

Hyperplasia of the spleen.

Pathologic Discussion

Dr. Tracy B. Mallory: This woman went through the very typical course of thrombopenic purpura with splenectomy. I do not believe Dr. Reynolds would want to guarantee that she would not have a recurrence of the disease, however. This is a disease that has aside from the multiple hemorrhages no anatomical picture. The spleens are not characteristic. They ordinarily show a rather marked degree of lymphoid hyperplasia but not necessarily. As far as I know, no one has ever recognized any diagnostic criteria by which the spleen of a case of purpura can be recognized microscopically, in contrast to hemolytic anemia where it is easy to recognize the situation. Likewise at autopsy, where it has been possible to study the bone marrow, nothing specific to the disease has been recognized. Even though there may be some diminution in megakaryocytes the findings again have been inconsistent. Cases do die from time to time and that is one definite reason for not putting off operation too long. I think the single most serious danger is that of cerebral hemorrhage which is by no means infrequent. We have four or five cases on record here with single or multiple cerebral hemorrhage as the immediate cause of death.

Dr. Vincent: It is only fair to say that the cerebral hemorrhages have all taken place after the spleen was out.

CASE 21382

Presentation of Case

A seven week old American male infant was admitted with a skin eruption of two weeks' duration.

The baby was apparently well at birth except for "sniffles" which persisted until admission. Two weeks before entry the mother noted a rash which began on the soles of the feet gradually involving the legs, thighs and face. The condition became progressively worse. In spite of the fact that the baby had never nursed very well because of the sniffles the mother believed that he had gained weight.

The father, two brothers and four sisters were living and well. When the mother was three months pregnant with the patient she had a rash on the palms of her hands, soles of her feet, thighs, genitalia and scalp. At this time a great deal of her hair fell out and she also had a sore throat. The rash and sore throat persisted for about four months.

Physical examination showed a poorly nourished but fairly well-developed infant. There was a maculopapular type of rash over both extremities from the soles of the feet to the upper thighs. There was much scaling over the soles. Around the nose and mouth the skin was fissured and crusted with some oozing. There were numerous rhonchi and râles throughout both chests, especially in the left lower lobe, but no signs of consolidation. The liver was felt two fingerbreadths below the costal margin in the right midclavicular line. The spleen was just palpable. There was some swelling of epiphysis of both radii.

The temperature was 100.8°, the pulse 130. The respirations were 25.

Examination of the blood showed a red cell count of 2,550,000, with a hemoglobin of 40 per cent. The white cell count was 45,600. Hinton and Wassermann tests were strongly positive.

Immediately after admission the baby received .05 grams of neoarsphenamin intravenously with no immediate reaction except for a temperature rise to 104° the following day. On the third day he developed a stiff neck. The Kernigs were positive. No signs of pneumonia could be demonstrated but he had many râles throughout the chest. He rapidly failed and died that day.

Differential Diagnosis

Dr. Richard S. Eustis: When I first saw this history, long before I reached the end of it, I had a diagnosis which I think you all have by now. It is an unusually typical history of congenital syphilis, with infection of the mother probable early in pregnancy. She had a perfectly definite case of lues which was transmitted to the fetus. There is no mention of whether the baby was full term or premature. The birth weight is not given so we cannot even make a guess from that as to whether it was premature, although the phrase "well developed" in the description of the physical examination at least indicates that the infant was not very premature.

The physical examination is very characteristic,—the eruption on the extremities, the scaling of the soles of the feet, the fissures and crusting around the nose and mouth. There is no mention of the anus. There might have been condylomata present there; presumably not, or they would have been mentioned. The liver was apparently slightly enlarged although not much so, as it is usually palpable in a baby of this age. Presumably the infant had some interstitial hepatitis due to syphilis. He also presumably has had an osteochondritis of the epiph-

yses, the long bones, showing up particularly in the radii. That is a very characteristic finding. What shall we make of the rhonchi and râles in the lung? I very much doubt if at this age, seven weeks, we have what is described as a white pneumonia, a syphilitic pneumonia. It seems much more likely that it is an extension of the process around the nose and mouth reaching down to the larynx and the trachea and giving us originally a syphilitic tracheitis and then secondary infection and probably bronchopneumonia, although on examination they could not find the spot or spots of consolidation.

The child had a definite secondary anemia undoubtedly due to the syphilis. The high white cell count is probably a polynuclear leucocytosis and probably indicates acute infection. There is no differential count reported.

At admission we have a seven week old infant with an active congenital syphilis. A sick infant because he is described as poorly nourished. The treatment he was given was perfectly proper. The only thing to do was to start treatment at once. I do not know whether they tried to get a dark field examination of any secretions from the fissures around the mouth. That probably would have confirmed their diagnosis. But in a situation like this, treatment should be started without waiting for a Wassermann test. The day following the injection of neoarsphenamin something happened to this infant. His temperature went up and shortly after the temperature went up he developed meningeal signs. The first thought that came to my mind was whether they were rash enough to give that injection into the longitudinal sinus. I regard that as a dangerous spot and I made inquiry about that and was told it went into the jugular so we cannot trace our meningeal symptoms to treatment that was meant for the longitudinal sinus and did not hit it. I do not think the injection of neoarsphenamin had anything to do with the rise in temperature, the meningeal symptoms and the death. I think it is much more likely that it is a terminal infection in an already sick infant with a secondary infection of the respiratory tract. There is no record of whether his respiratory rate went up at that time. That would help us to come to a conclusion. Meningeal signs occurring in the presence of an acute infection in an infant may mean meningitis, may mean otitis media, may mean "meningismus," and may mean, of course, any kind of meningitis.

In trying to sum up this picture without any information as to the state of the baby's ears, as to the appearance of the spinal fluid, knowing merely that it was a syphilitic infant who died very shortly after admission with a high temperature and with some stiffness of the neck and Kernig, it seems to me that the simplest diagnosis to make, one that will cover all the signs that we have described, is a diagnosis of congenital syphilis and probable bronchopneumonia. With the information we have we cannot exclude otitis media, and we cannot exclude a meningitis.

DR. HAROLD L. HIGGINS: About twenty-five per cent of congenital syphilitic babies have involvement of the central nervous system. We shall have to consider as a possible cause of death in this patient syphilis of the central nervous system, possibly syphilitic meningitis or syphilitic encephalitis.

Another very common cause of death in the syphilitic baby is respiratory obstruction; the respiratory obstruction is obstruction of the nose. The child has a rhinitis with a thick and possibly bloody discharge. The baby of seven weeks does not know how to breathe through his mouth and will not do so except when he cries. With his nose stopped up he will have difficulty inspiring, and may show as much retraction of the chest as a child with laryngeal diphtheria; when there is obstructed breathing in a child with a heart damaged by the spirochetes, myocardial failure and death of the patient may result. The râles in the chest may have been transmitted from the nose rather than be emanating from the bronchi or alveoli.

The syphilitic baby is likely to be premature and may present a nutritional problem. It is quite important that the baby receive proper nourishment and as good a feeding as one can give; this is best accomplished through the breast milk. One problem that came up with this baby was the question of keeping it with the mother so that the mother would be able to nurse the baby.

The giving of the arsphenamin may have stirred up the syphilis, especially the cerebral syphilis; as the rise in body temperature suggests. However, I still think that the treatment that was given the child was the correct treatment. One or two injections of neoarsphenamin given to a child like this will usually clear up the symptoms surprisingly. In a great many cases after a few days one may not recognize the child as sick. The rash disappears, the snuffles clear up, and the patient seems very much better. I do not see how in this case we can decide on the data given whether the death was from cerebral syphilis with damage to vital cells in the brain.

DR. AUSTIN W. CHEEVER: I think the situation has been very well covered. I certainly think that there was ample evidence on which to make a diagnosis without waiting for blood tests and to start treatment immediately. I am inclined to believe that the death was a Herxheimer reaction but I cannot be at all sure. If so, I do not think one should be disturbed about it because if the child was in such bad condition that one injection of neoarsphenamin would light up the Herxheimer seriously enough to cause death, in all probability if milder treat-

ment had been started, such as mercury, in the hope of preventing a Herxheimer reaction, the child would have grown rapidly worse from the syphilis and would probably have died from lack of sufficiently energetic treatment. The Herxheimer reaction in adults with late syphilis is something to be watched for and avoided if possible. But that is a different problem. With syphilis in the adult it is not so overwhelming as it usually is in babies. I feel this was a death from syphilis probably all the way through.

DR. HIGGINS: Do you not think it is rather unusual that the symptoms of what we may call secondary syphilis appeared when the child was five weeks old instead of being present when the child was born?

DR. CHEEVER: No, I do not. I rarely have seen any evidence younger than two or three weeks. The books give one the idea that the child is frequently born with early syphilis and has the wizened old man look, but I have seen only one or two. As you look back I think you have not seen very many. I think from two to three weeks is about the time these rashes start.

CLINICAL DIAGNOSIS

Congenital syphilis.

DR. RICHARD S. EUSTIS'S DIAGNOSES

Congenital syphilis.
Bronchopneumonia.
Meningitis?

ANATOMIC DIAGNOSES

Congenital syphilis.
Bronchopneumonia.
Luetic meningitis?
Cirrhosis of the liver, syphilitic.
Fibrosis of the adrenal cortex.
Septicemia, streptococcus hemolyticus.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: I think the postmortem can only answer a few questions, certainly not all the interesting ones in this case. There is no doubt that the child had congenital syphilis, of course. The most striking features at autopsy, confirming what was found on clinical examination, were the conditions of the liver which showed diffuse cirrhosis of the type that is found only in congenital syphilis, and a uniform fibrosis running throughout the lobules. The epiphyseal lines showed very slight changes. I would not be able to make a flat-footed diagnosis from them alone, but on the basis of the liver there can be no question that we are dealing with congenital syphilis. The autopsy was restricted and we did not examine the head. A postmortem lumbar puncture was done and that gave a fairly turbid fluid with some increase in lymphocytes. A Wassermann was done and was anticomplementary.

I do not know any way of making a postmortem diagnosis of a Herxheimer reaction. The child did have a fairly extensive bronchopneumonia, both upper lobes were consolidated and the sections look histologically like a perfectly ordinary bronchopneumonia of the secondary infectious type. The blood culture showed hemolytic streptococcus. It certainly was not a syphilitic pneumonia.

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ACUTE LYMPHOCYTIC MENINGITIS

FIRST clearly described by Wallgren in children and by Viets and Watts in adults, a syndrome, variously called "aseptic", idiopathic, epidemic and acute lymphocytic meningitis, has been thought by some to be a clinical entity, in spite of its unknown etiology. The disease, self-limited in character, with a mild lymphocytic formula in the cerebrospinal fluid showed many constant signs that suggests a single causative agent. Only recently, as the result of Armstrong's investigations in the laboratory of the National Institute of Health, United States Public Health Service, the probable etiology has been disclosed.

The cause of this malady is a filtrable virus, recovered by Armstrong and Lillie and reported by them in 1934. The disease has not only been developed in monkeys but protective antibodies have been found in the blood serum of recovered human cases, both by Armstrong and by Rivers and Scott.

It is therefore felt that the symptom complex is a disease entity, that the etiology is a filtrable virus and that the term "aseptic", as used by Viets and Watts, ought, in the light of more recent knowledge, to be replaced by another designation such as the one suggested by Armstrong, "acute lymphocytic choriomeningitis", or, more simply, "acute lymphocytic meningitis".

If the work of Armstrong and the co-workers is upheld, as it appears to be by the report of others, a new disease should be added to medicine. This suggestion, made first by Wallgren in 1926 and again by Viets and Watts in 1929 and 1934, is now made more certain by the finding of a filtrable virus as the etiological agent.

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4. Rivers, T. M., and Scott, T. F. M.: Meningitis in man caused by a filterable virus. Science **81:** 439 (May 3) 1935.
5. Viets, Henry R., and Watts, James W.: Aseptic (lymphocytic) meningitis. J. A. M. A. **93:** 1553 (Nov. 16) 1929.
6. Viets, Henry R., and Watts, James W.: Acute aseptic meningitis. J. Nerv. & Ment. Dis. **80:** 253 (Sept.) 1934.
7. Wallgren, A.: Peculiar form of epidemic meningitis (acute, "aseptic" meningitis). Wien. Arch. f. inn. Med. **12:** 297 (Feb.) 1926.

CANINE DAYS

ONE did not have to sense the heat or to read the reports of drowning and week-end motor casualties to realize that we have passed through another summer. A glance through our public prints told us that it was the silly season in newspaperdom. We know that that is the time of year when space-fillers are required, so we do not object to the trivial and banal notices that round out many a column. We pass over with a deprecatory smile accounts of "human icicles" and their mock heroism in subjecting themselves to bizarre experiments, although we frown a little when such publicity exploits are described as "aids to science". We wonder what specialty is practiced by the "special correspondents" who by cable would startle the world by telling us that the scientists assembled in Moscow have at last discovered that cretinism and some nervous disorders are due to dysfunction of the thyroid gland. Those uninformed readers who like this sort of mental pabulum are welcome to it.

Our objection is directed not against these cheap and stale items but against the avidity with which the agents of the large press services hunt out and report untried remedies and unproved medical theories. We object not because our complacency is disturbed but because

of the lamentable and tragic sequels of such stories.

We remember with sorrow the despair that followed the highly exploited visit of a Berlin physician, who, a large advertising syndicate would have us believe, held in his notorious "turtle bacillus" a cure for tuberculous infections. We recall the renewed hopes of tuberculosis sufferers at this announcement, how they deserted their physicians, their sanatoria and health resorts, only to return poorer in pocket and in health, with hopes crushed and frequently with cold abscesses for full measure. It was an outrageous affair.

Only recently we read in our esteemed *New York Herald Tribune** an account with the arresting headline, "Cancer Arrest by New Liquid Product". It related how an Ontario physician had, from harmless tiny organisms, produced a preparation which arrested the development of "carcinoma cancer". We know of the wave of eagerness which must have swept over the ranks of cancer victims when they read this "news", we know of their rush to secure some of this precious "Enesol", and, unless all precedents fail, we can foresee the cruel disappointment which may await them. If they lost faith in the medical news of their daily newspaper we would applaud, but, instead, they lose faith in medical science.

Our reproach is not directed against the Canadian doctor who gave in to the insistence of newsmongers, although it is a human possibility, if not a modern human tendency, to resist high-pressure reporters and their cameramen. Our sympathy goes to him. Our resentment is directed against the large news-gathering associations that serve our newspapers, and against the editors who not only accept these premature reports but give them bold display in their sheets. There is the "Science News Service" which gives to those publishers who desire it reliable accounts of scientific progress, but these accounts are not sufficiently florid to satisfy the present-day craving for "hot" news. The New York Academy of Medicine offers the daily papers wise estimates of medical events and discoveries. In its conservative comment (by Doctor Goldstein) on Doctor Connell's reported cure, he sounded the warning that "bitter experience has taught us to accept all announcements of discoveries on the cause or cure of cancer with utmost caution". The *Herald-Tribune* in its caption "Academy of Medicine Here Calls Report Remarkable," omitted Doctor Goldstein's "if correct". It would have pulled their punch.

We have had previous occasion to inveigh against the unscrupulous newspaper advertisement of nostrums, and now, for the public's sake and in the interest of the practice of medicine, we beg for expert supervision over the stories of medical affairs that go into our newspapers. We cannot rid our memories of the tragedies that have been caused by this cruel exploitation of alleged cures.

*Issue of July 18, 1935.

PHYSICIANS AND VITAL STATISTICS

To the physician in his practice, vital statistics seem remote, and since, in Massachusetts they are collated for the Commonwealth by the Secretary of State and not by the Commissioner of Public Health, they may appear almost irrelevant. He forgets that among other things they give a picture of what he is doing.

It may not be of general interest that last year one person died in each of the towns of Gosnold, Middlefield and Rowe or that even six were so recorded in Gill, but the charts which appear in the Annual Report should be studied not only by every physician, but by every person interested in the health of the people of the Commonwealth. There are shown graphically the rapid increase since 1930 of the number of deaths from heart disease and from cancer and from diabetes, a continuing upward swing in these figures. The appalling number of deaths from automobile accidents rose sharply in 1934. There is a little improvement in dealing with appendicitis, the mortality from which one would expect to find diminishing instead of increasing as it has in recent years. The figures for tuberculosis and for "infant mortality" are the most encouraging.

This statistical presentation is fascinating in spite of the well-known inaccuracy of the method because in the case of each group it makes insistent for the thoughtful physician the question "Why?" The multiplicity of challenges is a little disconcerting.

There are two obvious errors associated with the statistical method: errors of fact and errors of interpretation. Concerning errors of fact the Report says: "The chief element of error in mortality statistics lies in the inaccuracy of the statement of the cause of death as reported by the attending physician", and the mitigation of the severity of this condemnation follows: "who may not have been in a position to feel reasonably certain as to the nature of the terminal illness".

It is not that the physician has any intent to deceive. Too often he is unaware, in the nature of things, of the exact changes which have taken place in the body of his patient and his statement of the cause of death is an approximation toward a final diagnosis. It behooves every physician, in the interest of science, that pole star of practice, to use every effort to make his approximation as close as possible to the actual facts, and be not satisfied with merely meeting the minimum requirements under which a death certificate will be accepted by the town clerk.

THE COMMONHEALTH

Tuberculosis

A valuable issue of the *Commonhealth* (Vol. 22, No. 1), the quarterly bulletin of the Massachusetts Department of Public Health, is devoted to the subject of Tuberculosis and is made up of articles by eminent authorities on such subjects as Tuberculosis in the Practice of Medicine, The Differential Diagnosis of Tuberculosis, The Diagnostic Dispensary, The Cure of Tuberculosis, The Rôle of Surgery in Pulmonary Tuberculosis, Hospitalization of Tuberculosis in Massachusetts, Diet in the Home Treatment of Tuberculosis, Extra-Pulmonary Tuberculosis, Tuberculosis in Children, The School Tuberculosis Clinic Program, Chadwick Clinic Results, Tuberculosis Case-Finding in Children of School Age, Summer Health Camps, Teaching the Etiology of Tuberculosis, Policies and Routines for a Tuberculosis Nursing Service and Social Work for the Tuberculosis Patient. An article by Dr. Alton S. Pope, Director of the Division of Tuberculosis, shows the decline in the death rate from pulmonary tuberculosis from approximately 450 for females and 324 for males in 1849 to approximately 40 for females and 60 for males in 1933.

Certain definite advances have been made in recent years in the attack on tuberculosis. In the treatment of the adult type of pulmonary tuberculosis, collapse therapy has become widely accepted and has been responsible for the saving of many lives, and the shortening of the course of the disease in innumerable instances. Our new conception of the distinction between primary tuberculosis as it occurs in the child, in the non-sensitive individual, and reinfection giving rise to the adult type in the sensitized individual, either adult or child, has clarified the picture of the course of the disease and the part that allergy plays in that picture. We realize now that childhood tuberculosis, important as it is, is fundamentally a benign infection, but that, instead of immunizing the individual, as was formerly believed, it actually renders his tissues susceptible to the tubercle bacillus so that a reinfection will give rise to the adult type of phthisis.

In Massachusetts, particularly, a fundamental concept in tuberculosis control has been put into effect with the Ten Year Program of case-finding in the public schools of the state. In this ten years, just ended, 400,000 school children have been examined by tuberculin test and x-ray of reactors and the work will continue under the control of the regional sanatoria. In Massachusetts, also, the tuberculosis summer health camp has reached its fullest development under the joint aegis of the tuberculosis associations and the county sanatoria, ever mindful of the doctrine that "a camp is as good as its follow-up."

The treatment of tuberculosis must continue, but in prevention lies the way to its eradication, and prevention depends on case-finding and follow-up.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Munro, Donald. A.B., M.D. Harvard University Medical School 1916. F.A.C.S. Assistant Professor of Neurological Surgery, Harvard Medical School. Surgeon in charge of the Neurosurgical Service, Boston City Hospital, Boston. His subject is "The Treatment of Compound Fracture of the Skull." Page 551. Address: 818 Harrison Avenue, Boston.

Bowman, Karl M. A.B., M.D. University of California 1913. Assistant Physician, Bloomingdale Hospital 1915-1921. Chief Medical Officer, Boston Psychopathic Hospital 1921-. Assistant Professor of Clinical Psychiatry, Boston University School of Medicine. Special Instructor in Social Psychiatry, Simmons College School of Social Work. Address: 74 Fenwood Road, Boston, Mass. Associated with him is

Howard, Paul M. B.A., M.D. University of Pennsylvania School of Medicine 1933. Interne, Methodist Episcopal Hospital, Philadelphia, Pa., 1933-1934. Interne, Boston Psychopathic Hospital, 1934-1935. Interne, Neurological Service, Boston City Hospital, 1935. Address: Boston City Hospital, Boston. Their subject is "Report of a Case of Lead Encephalitis Due to Tri-Ethyl Lead." Page 559.

Barbour, Ellen H. Graduate, Simmons College School of Social Work. Social Worker at Clinic for "Study of Growth Development of Normal Child," Harvard School of Public Health. Her subject is "Adjustments During Four Years of Patients Handicapped by Poliomyelitis." Page 563. Address: 54 Hancock Street, Lexington, Mass.

Marks, George A. A.B., M.D. Harvard University Medical School 1928. Assistant in Surgery, Massachusetts General Hospital. Junior Visiting Surgeon, Chelsea Memorial Hospital. Surgical Assistant, Huntington Memorial Hospital. Assistant in Surgery, Tufts College Medical School, and the Cambridge Hospital. His subject is "A Method of Repair of Femoral Herniae by a Fascial Strip from the Aponeurosis of the External Oblique Muscle." Page 565. Address: 198 Commonwealth Avenue, Boston.

McGill, Chester F. M.D. Tufts College Medical School 1915. His subject is "Congenital Pyloric Obstruction." Page 567. Address: 82 Congress Street, Portsmouth, New Hampshire.

SCHALL, LEROY A. M.D. Jefferson Medical College of Philadelphia 1917. F.A.C.S. Instructor in Laryngology, Harvard University Medical School. Surgeon, Massachusetts Eye and Ear Infirmary. Assistant Laryngologist, Huntington Memorial, Palmer Memorial and Robert Breck Brigham Hospitals. His subject is "Progress in Laryngology." Page 574. Address: 270 Commonwealth Avenue, Boston.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. KICKHAM, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.
R. S. TITUS, M.D., *Secretary*, 472 Commonwealth Ave., Boston, Mass.

THE ANEMIAS OF PREGNANCY

Thanks to the excellent work of Strauss, Castle and others, the hitherto vague subject of anemia in pregnancy has been clarified, its etiological factors ascertained and correlated, and the treatment made sure and simple. Recent studies have shown that a large percentage of pregnant women show a varying degree of anemia from no apparent cause, i.e., in the absence of hemorrhage, sepsis or chronic disease. These "anemias of pregnancy" are divided into two types: The hypochromic anemia (corresponding to simple secondary anemia), and the macrocytic anemia of pregnancy (corresponding to pernicious or Addisonian anemia). It should be noted here that this is not the true pernicious anemia and differs from it in the fact that it clears up after pregnancy is terminated and does not recur.

Hypochromic anemia has its etiology in several factors: (1) defective diet, principally a lack of sufficient iron; (2) disturbances in gastric secretion, i.e., a lowering or absence of free hydrochloric acid. This gastric inefficiency has been shown to occur in more than fifty per cent of all pregnant women. (3) Inasmuch as the fetus draws its blood-forming materials from the mother, this represents a "chronic blood loss" to the mother.

The blood picture in these cases is that of simple secondary anemia; the decrease in hemoglobin is greater than the decrease in the number of red blood cells. The patient shows varying symptoms, pallor, fatigue, edema, dyspnea, digestive disturbances, etc.

The administration of iron in the form of iron and ammonium citrate, ninety grains a day, or of ferrous sulphate, nine grains a day, will cure this condition and restore the patient to normal health.

Macrocytic anemia of pregnancy is fortunately not so common. This is frequently referred to as the primary anemia of pregnancy. It has its etiology in similar factors: (1). Dietary insufficiency (principally a lack of animal protein [vitamins] B). (2). Gastric insufficiency or lack of a specific factor in the gastric secretion necessary for the formation of blood cells. Experiment has shown that both these factors are essential for normal hematopoiesis.

The blood picture in these cases is like that of pernicious anemia. The decrease in the number of red cells is greater, proportionately, than the decrease in hemoglobin content, giving a color index of +1. Macrocytes and microcytes abound. The symptoms are more severe, weakness, nausea, vomiting, diarrhea, sore tongue, etc.

Liver or liver extract by mouth or injection will cure these patients. Relapse does not occur. Inasmuch as these patients have the same deficiencies that cause hypochromic anemia, iron must be added to the treatment.

As a matter of prevention, the diets of pregnant mothers should be sufficiently rich in iron and animal protein. Small doses of iron in either of the forms mentioned should be given prophylactically to every patient.

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

MISCELLANY

DR. GEORGE BURGESS MAGRATH HAS RESIGNED

The resignation of Dr. George Burgess Magrath, for many years medical examiner of Suffolk County, has been announced.

Dr. Magrath has occupied an enviable position in the confidence of the medical profession and the courts not only in Massachusetts but throughout the country, for he has played a prominent part in the development of the Medical Examiner System in supplanting the old coroner practice.

His reputation for profound knowledge of the problems incident to homicide has led to his employment in many prominent trials. Like many eminent persons in public life, he has survived the opposition of those who on occasion have tried to prevent his reappointment. A long list of governors has continued Dr. Magrath in office, which is a tribute to his standing.

It is hoped that he will have the comfort and enjoyment of a serene release from the controversies of public life. The *Journal* congratulates Dr. Magrath on his honorable retirement.

ANTERIOR POLIOMYELITIS CASES FOR 1935

WEEKLY LIST, SEPTEMBER 9 TO 14

City or Town	
Dartmouth	1
Fall River	6

Lakeville	1
Somerset	2
Taunton	2
Westport	1
Brockton	2
Dedham	1
Franklin	2
Milton	1
Quincy	1
Weymouth	2
Boston	56
Brookline	1
Cambridge	6
Chelsea	1
Everett	3
Lexington	1
Malden	5
Medford	3
Melrose	1
Newton	3
Somerville	6
Watertown	1
Winchester	1
Andover	1
Beverly	1
Gloucester	1
Groveland	1
Haverhill	7
Lowell	3
Lynn	2
Newburyport	1
North Andover	1
Reading	2
Salem	3
Fitchburg	1
Leominster	1
Milford	1
Westminster	1
Sterling	1
Springfield	2
Dalton	1
Hinsdale	1
Pittsfield	1
TOTAL	143

APPOINTMENT OF DR. J. J. GOODWIN

Governor Curley nominated and the Council confirmed the reappointment of Dr. J. J. Goodwin to the position of Medical Examiner of the Fourth Worcester District.

A LECTURE BY DR. CHARLES F. WILINSKY

Dr. Charles F. Wilinsky, Deputy Health Commissioner of Boston and Director of the Beth Israel Hospital, lectured September 12-13 on "Public Health and Community Relations" at the Annual Institute for Hospital Administrators being held in Chicago September 11-25, 1935. This Institute is sponsored by the American Hospital Association, the University of Chicago and the Chicago Hospital Association and is being conducted for the purpose of furthering the training of hospital superintendents.

DIAGNOSIS IN AN EPITAPH

BY FREDERICK C. WAITE

One finds unexpected novelties in the epitaphs of nineteenth century tombstones. Usually they have to do with the uncertainty of life and spiritual preparation for what follows. The epitaph here quoted, probably written by a physician, has a quite different purpose, that of medical advice to the living.

The physician mentioned, Dr. Charles Knowlton (1800-1850), was a graduate of Dartmouth Medical College in 1824 and also received an honorary degree of doctor of medicine from Berkshire Medical Institution at Pittsfield, Massachusetts, in 1827. He was a member of the Massachusetts Medical Society from 1844 and practiced at Ashfield, Massachusetts, some thirty-five miles from Pittsfield, for twenty-five years.

In the cemetery at Ashfield today stands a stone with the following inscription:

Stephen Owen
son of Dr. C. Knowlton
died
Sept. 11 1849
ae. 20 yrs. 10 mo. 12 d.
Reader: If your friend breath
too frequently however mild the
other symptoms rest not easy
under the fallacious idea that
"It is nothing but a cold."

One may infer that this son was the victim of pneumonia, and that the epitaph aimed to prevent a similar catastrophe among his friends and neighbors. Since the father died five months after this son's death it is possible that the epitaph was written by his surviving brother, Dr. Charles Lorenzo Knowlton (1824-1898), who attended at Berkshire Medical Institution in 1843-44 and graduated at Jefferson Medical college in 1845. He practiced for over fifty years, the greater part of this time at Northampton, Massachusetts. He was a member of the Massachusetts Medical Society from 1851.

OUTING AT THE WOLLASTON GOLF CLUB

A joint golf outing of the Quincy City Hospital Staff, the Norfolk South District Medical Society and the South Shore Dental Society was held at the Wollaston Golf Club on Wednesday, Sept. 4, 1935.

About forty of the combined group played golf under inclement conditions.

The outing was successful in a "get-together" as the foursomes were arranged to include both medical and dental men.

DR. BURWELL'S POSITION ON THE STAFF OF THE PETER BENT BRIGHAM HOSPITAL

Dr. Charles S. Burwell, Dean of the Harvard University Medical School, has been appointed to the position of Physician on the Staff of the Peter Bent Brigham Hospital.

RECENT DEATHS

GOODELL—George Zina Goodell, M.D., of 5 Williams Street, Salem, Massachusetts, died at his home September 9 after a long illness. He retired from practice several years ago. Dr. Goodell was born in Salem in 1859 and after graduation from the high school studied at and graduated from the Harvard University Medical School in 1881.

He joined the Massachusetts Medical Society in 1881 and retired in 1933. He devoted his major activities to diseases of the ear, nose and throat. He served on the staff of the Salem Hospital from 1883 to 1894 and in 1889 and 1890 was Superintendent of that institution.

He was treasurer of the Essex South District Medical Society for twenty-five years and was a member of the New England Society for Physical Therapeutics, Essex Lodge of Odd Fellows and the local lodge of United Workmen.

Dr. Goodell is survived by his daughter, Mrs. Raymond H. Odell; two brothers, Frank T. and Oliver W. Goodell; and two sisters, Mrs. George E. Patterson and Mrs. Walter P. Pratt, all of Salem.

HALE—Edward P. Hale, M.D., of Lenox, Massachusetts, aged seventy-five, died at his home September 9, 1935. Dr. Hale was born in Danvers, Massachusetts, and graduated from the Bellevue Hospital Medical College in 1881. He practiced in Vermont and also was resident physician at the Danvers State Hospital for a brief period before settling in Lenox.

He was president of the Lenox Savings Bank, a trustee of the Lenox Academy and a member of the Massachusetts Medical and local Societies.

Dr. Hale is survived by his widow, Mrs. Anna P. (Walker) Hale.

CRONIN—Thomas Joseph Cronin, M.D., of 29 Forest Street, with an office at 36 Pleasant Street, Worcester, Massachusetts, died at the Memorial Hospital, Worcester, September 10, 1935.

Dr. Cronin was born in 1865 at Braidwood, Illinois, the son of Mr. and Mrs. Henry D. Cronin. Early in his life the family moved to Millbury, Massachusetts, where his preliminary education was acquired; later he attended the College of Pharmacy in Boston. He graduated in medicine from the Baltimore University School of Medicine in 1898. Before settling in Worcester, he practiced for several years in Webster, Massachusetts. For many years he was eye, ear, nose and throat consultant at the Fairlawn Hospital. He joined the Massachusetts Medical Society in 1898 and was also a Fellow of the American Medical Association.

Dr. Cronin was prominent in Masonic circles, the Worcester Economic Club and the Central Congregational Church, Worcester. He is survived by his widow, Mrs. Louise (Schwegler) Cronin, two daughters, Miss Dorothy Cronin and Mrs. Frank L. Harrington, a brother, John D. Cronin, and a sister, Mrs. Nellie Feehan, both of Millbury.

NOTICES

IMPORTANT ANNOUNCEMENT TO PHYSICIANS AND HOSPITALS

Due to physicians' unfamiliarity with Government requirements in submitting charges, more than 50,000 accounts submitted by physicians and hospitals in Civil Works Administration injury cases were returned for correction, or held for further information, necessitating undue delay in payment, because of one or more discrepancies.

As a former supervising auditor of the U. S. Employees' Compensation Commission, I have condensed to a four page booklet under the title "Instructions to Physicians and Hospitals for Submitting Charges in Federal Injury Cases," the numerous requirements scattered throughout 60 detailed pages of the Government's regulations, each point being separately paragraphed and headed for the most convenient reference.

The following points are covered:

- Authority for treatment
- Medical and x-ray reports
- X-ray films
- Charges in disease cases
- Postmortem examinations
- Dental charges
- Charges for drugs
- Charges for appliances
- Physicians owning hospitals
- Payment for personal property
- Submission of x-ray charges
- Charges for blood
- Itemization of accounts
- When to submit charges
- Joint accounts
- Required signatures
- Hospital charges
- Nurses' charges
- Assistants' and consultants' services

In addition to the foregoing the booklet covers several important points such as inquiry as to payment of accounts, time required for receipt of remittances after payment is authorized, how charges may be expedited for payment, and other valuable information to physicians and hospitals having charge in Federal injury cases.

This booklet is neatly prepared, has a durable cover, and is convenient for pocket or office use.

It is an accurate and ready reference, covering every requirement, a guide to coöperation and the avenue to correct submission and the earliest possible payment of accounts. This guide will save time, money, worry, and waiting.

With an increasing number of people being employed subject to Government compensation benefits, and with the near possibility of an added number of physicians being called upon to serve their Government as a result of recent social legislation, every physician should familiarize himself with Government requirements in respect to charges.

Price: $1.00 the copy, postpaid. The price is negligible in comparison with the booklet's practical and beneficial value. Order now.

C. B. RIDDLE.

Washington, D. C., P. O. Box 442.

DR. WILLIAM P. MURPHY ASSUMES INDEPENDENT PRACTICE

Dr. William P. Murphy, formerly associated with Dr. Edwin A. Locke and Dr. George R. Minot, will continue his office and consultation practice independently at his present address, 311 Beacon Street, Boston, Massachusetts.

DR. S. F. MARSHALL JOINS THE LAHEY CLINIC

Dr. Frank H. Lahey wishes to announce that Dr. Samuel Frederick Marshall, who for the past ten years has been associated in surgery with the Henry Ford Hospital in Detroit, Michigan, has been appointed as one of the surgeons to the Lahey Clinic.

WORCESTER NORTH CANCER CLINIC
Burbank Hospital
Fitchburg, Massachusetts

NOTICE TO MEMBERS OF THE WORCESTER NORTH DISTRICT MEDICAL SOCIETY

September 11, 1935.

My dear Doctor:

There will be a consultation cancer clinic September 24 at the Burbank Hospital; 9:30 a.m. to 12:30 p.m. The consultants are: Dr. Grantley W. Taylor, visiting surgeon to the Massachusetts General, Huntington Memorial, and Pondville Hospitals, and Dr. Charles C. Lund, visiting surgeon to the Boston City and Huntington Hospitals.

We hope you will use this opportunity to be present with or refer by note any of your patients having cancer or suspicious of a malignant condition.

In order to facilitate the clinical routine, we would appreciate the patients' arrival at 9 o'clock. It has proved in other consultation clinics of value to have available any history regarding the patients' condition. For your convenience in this matter, we would be very glad to have you telephone at any time Fitchburg 4807 to give information to the clinic clerk, Miss Irma Holmes.

We will appreciate your active participation with the clinic.

Very truly yours,

WORCESTER NORTH CANCER COMMITTEE
DR. F. H. THOMPSON, SR., *Chr.*
DR. WALTER F. SAWYER, *Secy.*
DR. HERVEY B. PITCHER
DR. ERSKINE R. PICKWICK
DR. CHARLES J. LASERTE.

ANNOUNCEMENT

CLARK W. HEATH, M.D., has opened an office at 319 Longwood Avenue, Boston, Mass.

REPORT AND NOTICE OF MEETINGS

THE FOUR COUNTY MEDICAL SOCIETY

An interesting day was spent by the men who attended the meeting of the Four County Medical Society in Greenfield, on Wednesday, September 11. A series of case presentations was made by Greenfield doctors at the Franklin County Hospital and the following papers were presented:

Hyperthyroidism, Dr. F. B. Sweet, of Springfield.

Some Problems in Gall Bladder Surgery, Dr. I. S. F. Dodd, of Pittsfield.

Some Observations on the Use of Evipal and Avertin, by Dr. E. A. Knowlton, of Holyoke.

Femoral Hernia—The Inguinal Approach, by Dr. F. Hagler, of Springfield.

The State Pneumonia Program, by Dr. R. Heffron, of Boston.

The President of the Massachusetts Medical Society, Dr. Charles Mongan, addressed the gathering in his usual pleasing manner. He stressed the importance of public-minded physicians and outlined the transactions taking place in Washington, relative to the Social Security Law. Doctor Mongan placed especial emphasis on the need of coöperation and felt that we could best use our abilities in educating and molding public opinion on matters of socialized medicine.

An excellent luncheon was served at the Hotel Weldon and many of the doctors attending took advantage of the invitation of the Greenfield Golf Club to play on its links during the afternoon.

J. A. WHITNEY, *Secretary.*

THE CARNEY HOSPITAL

CLINICAL MEETING

September 23, 1935, 8 P.M.

The Clinical Diagnosis of Jaundice. Dr. Howard M. Clute. Lantern Slides.

The Technique of Arthroplasties. Dr. W. R. MacAusland. Motion Pictures.

Physicians and medical students invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, SEPTEMBER 23, 1935

Wednesday, September 25—

†12 M. Clinico-Pathological Conference. Children's Hospital.

Saturday, September 28—

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

September 23—The Carney Hospital, Clinical Meeting. See page 589.

September 24—Worcester North Cancer Clinic. See page 589.

September 27-28—New England Surgical Society. See page 549, issue of September 12.

September 30-October 12—International Medical Post-Graduate Courses in Berlin. For further information apply to the Geschäftsstelle der Berliner Akademie für ärtzliche Fortbildung, Robert Koch—Platz 7 (Kaiserin Friedrich-Haus), Berlin N. W. 7.

October 6-20—Seventh Annual Training Course For Medical Reservists at the Mayo Clinic. See page 441, issue of August 29.

October 7-10—American Public Health Association will meet in Milwaukee, Wisconsin. For information address the American Public Health Association, 50 West 50th Street, New York City. See page 573.

October 14-19—Inter-State Postgraduate Medical Association of North America. See page 549, issue of September 12.

October 21 - November 2—1935 Graduate Fortnight of the New York Academy of Medicine. See page 898, issue of May 9.

October 28 - November 1—The Twenty-Fifth Clinical Congress of the American College of Surgeons. See page 1065, issue of May 30.

BOOKS RECEIVED FOR REVIEW

Textbook of Attendant Nursing. Katharine Sheppard and Charles H. Lawrence. 433 pp. New York: The Macmillan Company. $3.00.

Aphasia. A clinical and psychological study. Theodore Weisenburg and Katharine E. McBride. 634 pp. New York: The Commonwealth Fund. $5.00.

Clinical Parasitology and Tropical Medicine. Dámaso de Rivas. 367 pp. Philadelphia: Lea & Febiger. $5.00.

I'd Live It Again. E. J. O'Meara. 324 pp. Philadelphia: J. B. Lippincott Company. $2.50.

International Clinics. Volume III. Forty-Fifth Series, 1935. Edited by Louis Hamman. 337 pp. Philadelphia, Montreal and London: J. B. Lippincott Company.

Diseases of the Liver, Gall Bladder, Ducts and Pancreas. Their diagnosis and treatment. Samuel Weiss. 1099 pp. New York: Paul B. Hoeber, Inc. $10.00.

Agents of Disease and Host Resistance. Including the principles of immunology, bacteriology, mycology, protozoölogy, parasitology and virus diseases. Frederick P. Gay. 1581 pp. Springfield and Baltimore: Charles C Thomas. $10.00.

Diseases of the Nervous System. A text-book of neurology and psychiatry. Smith Ely Jelliffe and William A. White. Sixth Edition, Thoroughly Revised. 1175 pp. Philadelphia: Lea & Febiger. $9.50.

Human Pathology. A Textbook. Howard T. Karsner. Fourth Edition, Revised. 1013 pp. Philadelphia and London. J. B. Lippincott Company.

The Doctor and the Public. A study of the sociology, economics, ethics, and philosophy of medicine, based on medical history. James Peter Warbasse. 572 pp. New York: Paul B. Hoeber, Inc. $5.00.

Clinical Diagnosis by Laboratory Methods. A working manual of clinical pathology. James Campbell Todd and Arthur Hawley Sanford. Eighth Edition, Thoroughly Revised. 792 pp. Philadelphia and London: W. B. Saunders Company. $6.00.

The Medicine-Man of the American Indian and His Cultural Background. William Thomas Corlett. 369 pp. Springfield and Baltimore: Charles C Thomas. $5.00.

BOOK REVIEWS

Surgical Diseases of the Chest. Evarts A. Graham, Jacob J. Singer, and Harry C. Ballon. 1070 pp. Philadelphia: Lea & Febiger. $15.00.

Advances in thoracic surgery during the past ten years have been swift-moving and spectacular. There are perhaps no more than a dozen clinics on this continent that have kept abreast of this rapid development or have made significant contributions to its progress. One of the foremost of these is the Barnes Hospital group under the leadership of Evarts Graham. A book from this source may be accepted as authoritative and trustworthy and any detailed review is superfluous.

There is a subtle distinction between a text on "surgical diseases of the chest" and one on "chest surgery." The former may be read by internists, the latter will not. The entire book has been designed to inform and stimulate interest in the reader, be he physician or surgeon. If an internist is not familiar with the facts presented in this book he should give up handling diseases of the chest.

The bibliography is a most exhaustive one and will give the edition lasting value. Obviously the text of such a work calls for revision before the ink is dry. No one appreciates this better, however, than the authors. The book is the product of the work of years, and the task of keeping such a voluminous manuscript open to revision up to the time of publication a trying one. The authors are to be congratulated on their success in this task, as the subject matter has been brought up to date to the moment of going to press.

The quality of the illustrations unfortunately is not in a class with the text. The reviewer has listed some 70 of the 637 illustrations that could well be omitted as they fail to show the points the authors wish emphasized, or are superfluous. In medical writing there is a fine distinction between illustrations used as proof for the statement of clinical or experimental facts and illustrations that clarify the text by providing the reader with a direct visual image. Too many examples of the former, even if they are well done, add to the bulk and expense of a book without adequate justification.

Personally, the reviewer would prefer a two

volume form, as hard usage has already shown the book too large and heavy for its binding.

The Principles and Practice of Urology. Frank Hinman. 1111 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

This monumental volume on the Principles and Practice of Urology is unquestionably one of the outstanding one volume contributions to Urology in years. One is amazed at the completeness, the scholarly presentation of the subject, and the extraordinary attention paid to details. There is no phase of Urology that is not thoroughly discussed.

The first part of the book is devoted to such subjects as the Comparative Anatomy of Urinary Excretion, of Reproduction, and of Urinary and Genital Association of the Urogenital Union. It is extremely well written and should prove of great value to the scientific urologic student.

Each urologic disease is discussed as to its etiology, pathology, differential diagnosis, and treatment. The book is full of illustrations, diagrams, and photomicrographs. Such recent urologic endeavors as the etiology of stone formation, the prostate-gonad-pituitary relationship, the ketogenic diet, and many others are brought up to date.

The volume reflects the energetic, indefatigable, studious character of the author. It is a classic and well worth the price of ten dollars.

Electrotherapy and Light Therapy. Richard Kovács. Second Edition. 696 pp. Philadelphia: Lea & Febiger. $7.50.

The major portion of this book presents a repetition of statements already published in other books on this subject and contained in the first edition. While a number of articles have been revised, in the main the essential factors remain the same. New chapters have been added on the subjects of hyperpyrexia, short wave and ultra short wave but so far, statements concerning the use of these measures are largely a matter of individual opinion; it will take observation of a large number of cases over a long time period before the value of these procedures will be known.

The book is rather too large for the purposes of ready reference but it would be of some value to a beginner in the field of physical therapy. Portions of the work which review what has been done by many well-known workers in this field may be found interesting and instructive to physicians who are following the various specialties in medicine and surgery.

The need for a thorough understanding of the basic principles involved in electrotherapy and the application of the technique is well emphasized. It is thoroughly indexed and the glossary which contains definitions authenticated by the American Medical Association should be helpful to the student.

Clinical Laboratory Methods and Diagnosis. A textbook on laboratory procedures with their interpretation. R. B. H. Gradwohl. 1028 pp. St. Louis: The C. V. Mosby Company. $8.50.

This comprehensive textbook on laboratory procedures covers the entire range from urine analysis to the performance of postmortem examinations, the preparation of museum specimens, and toxicological technique. Obviously to compass the entire field in a single volume there must be many omissions and much of the material must be slighted. In spite of this, a fairly adequate coverage is given by the present volume. The best section is that dealing with hematology. A good index facilitates the use of the volume. For Massachusetts physicians who have become thoroughly familiar with the usefulness of the Hinton test, it is a bit surprising that this has not been mentioned. A trained pathologist might take issue with some of the statements in the section on postmortem examinations, but to a person without special training called upon to do an occasional autopsy it is of some advantage. The typography is good but the coated paper is heavy, particularly for the binding, which raises some question as to how long it would hold together under the usual conditions of laboratory use.

Chronic Nasal Sinusitis and Its Relation to Mental Disorder. An applied pathology of abnormal conditions of the nasal sinuses found in mental hospital patients. F. A. Pickworth. 156 pp. London: H. K. Lewis & Co., Ltd. 16s net.

In a well-printed book of 150 pages with eighty-three illustrations of moderate excellence the author seeks to establish causal connection between nasal sinusitis and mental disorder of all forms. His opportunity for numberless postmortem examinations in a large institution for mental disease has led him to the following basic conclusions:

1. There is abundant historical evidence of an association between sinusitis and a great variety of mental disturbances.
2. Toxins and organisms can be vascularly and perivascularly transferred from infected sinuses to the brain.
3. Sinusitis can act as a cause of latent adjacent disease of cerebral capillaries.
4. Toxins from a contiguous nasal sinus can be directly absorbed intracranially.

Among the ten chapters are several which deal with subjects far afield from the main topic and which are quite adequately covered in any of the standard textbooks on sinus disease, such for example as anatomy and physiology, bacteriology, allergy, pathology and the relation of sinus disease to many general medical disorders. In the same manner several chapters dealing with the cerebral blood supply, the pathology of mental disorder, and the anatomy of the cranial nerves are reduplications of material to be found in any textbook of

nervous diseases. However in the latter chapter the author seeks to correlate these two subdivisions and by the use of photomicrographs and a few colored plates makes out an excellent and interesting case for the relationship between sinusitis and mental disease. The final chapter is devoted to a résumé of illustrative case histories, and there is an appendix describing the technique for preparation of gross and microscopic specimens. A long list of bibliographic references is appended.

The text is at times annoyingly replete with references to other authors, a feature which tends to break up both the author's and the reader's train of thought.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 93. 593 pp. New York: The Rockefeller Institute for Medical Research.

In this volume the experimental studies of Webster and his associates on the St. Louis encephalitis virus are continued. There is the usual wide range of material from the fields of organic chemistry, chemical pharmacology, physical chemistry, and general physiology. From the department of the hospital there are several papers of interest. Page and others present further work in the field of hypertension, and Page also has a report on the prevention of cholesterol-induced atherosclerosis in rabbits by feeding an organic iodine compound. Rivers presents a biological test for the diagnosis of psittacosis. The studies of Rhoads on various types of hematopoietic disturbances are continued.

Apparatus and Technique for Roentgenography of the Chest. 1935. Charles Weyl and S. Reid Warren, Jr. 166 pp. Springfield and Baltimore: Charles C Thomas. $5.00.

This volume of 166 pages is divided into four chapters and an appendix.

Chapter one is a summary of the phenomena associated with the production of chest roentgenograms. Chapter two is devoted to a discussion of radiographic apparatus including not only that necessary for the actual taking of films but also the various devices used for the dark-room technique. The technical procedures for the taking of films of the chest and their development are covered in detail in chapter three. In chapter four the physical measurements for determining, and apparatus used, for the output of x-ray machines and the quality of x-ray films are described. In the appendix the elementary electrical theories are discussed.

The material is so arranged that reference is easy. There are several diagrams and valuable tables. The book is designed for the roentgenologist rather than the technician.

The authors believe, and your reviewer agrees with them, that a thorough understanding of the physical principles underlying the making and processing of x-ray films of the chest is a necessary part of roentgenological training. For the lay-technician the book is perhaps a little too theoretical and the data for guidance in individual exposures lacking.

Six Conférences de Physiologie. Leon Binet. 73 pp. Paris: Masson et Cie. 12 fr.

This booklet assembles the contributions made in physiology during the last few years in the laboratory of physiology of Professor Binet. It includes experiments on the lungs, the suprarenal capsules, nerve tissue, induced hyperthermy and on the use of fish both in physiology and in toxicologic assay. These conferences should be of interest and value to the student of physiology.

Oxford Medical Publications. Gastritis and Its Consequences. Knud Faber. 119 pp. London: Oxford University Press. $3.00.

This excellent monograph is comprised of the three Guy's Hospital Medical School lectures delivered by the author in November, 1934. The subject material covers the extensive field from the early pathological studies of the author through the very recent concepts of the relation of gastritis to polyposis, pernicious anemia, and gastric cancer.

Faber frankly acknowledges the "stamp of his own personal views", but he has supported his opinions not only with well-chosen pathological data and logical analysis, but also with contributory data from other workers. The conclusions are neither dogmatic nor biased, and the whole tenor of the book is stimulating and thought-provoking. It is well worth reading with great care, even though one cannot accept it entirely without reservations.

Docteur Carlos J. Finlay. Son centenaire 1933. Sa Decouverte 1881. Francisco Dominguez. 302 pp. Paris: Librairie Louis Arnette.

The name of Doctor Carlos Finlay is unfortunately only too little known in the United States. Although his medical education and early training were obtained in this country, his many important medical contributions were accomplished in Cuba, where he was considered a genius. Finlay's studies of yellow fever were epoch-making and practically laid the entire basis for Cuba's present-day sanitation system. In fact his biographer considers his discoveries as the greatest made in America in the nineteenth century.

Confined primarily to extensive and minute studies of the various phases of yellow fever, his versatility extended, however, to other medical fields. Exophthalmic goitre, cholera, tetanus, electrotherapy, leprosy filariasis, trichiniasis, beriberi, tuberculosis and even ophthalmology (to which he contributed a new operation for cataract) received his studied attention. The present volume which is published through the efforts of the Cuban government under the auspices of the Finlay Centenary Committee, reviews his more important work and recounts the highlights of his career.